Glypressin Experience before Liver Transplantation

中國醫藥大學附設醫院

消化系內科

賴 學 洲

2013/08/22

OUTLINE

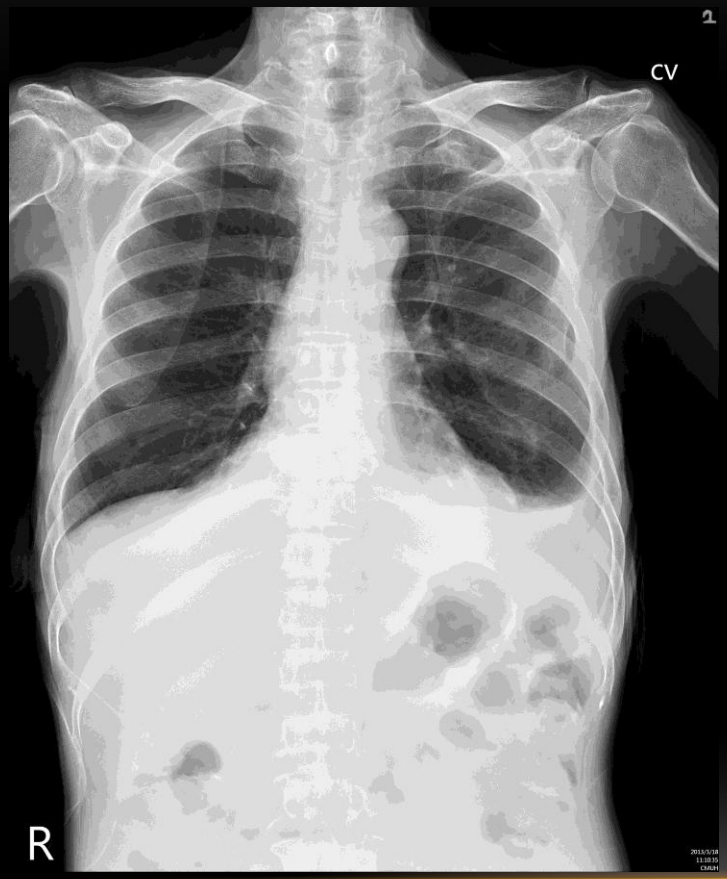
- Case presentation
- Cirrhosis scoring system
- Impaction of hepatorenal syndrome on liver transplant
- Conclusion

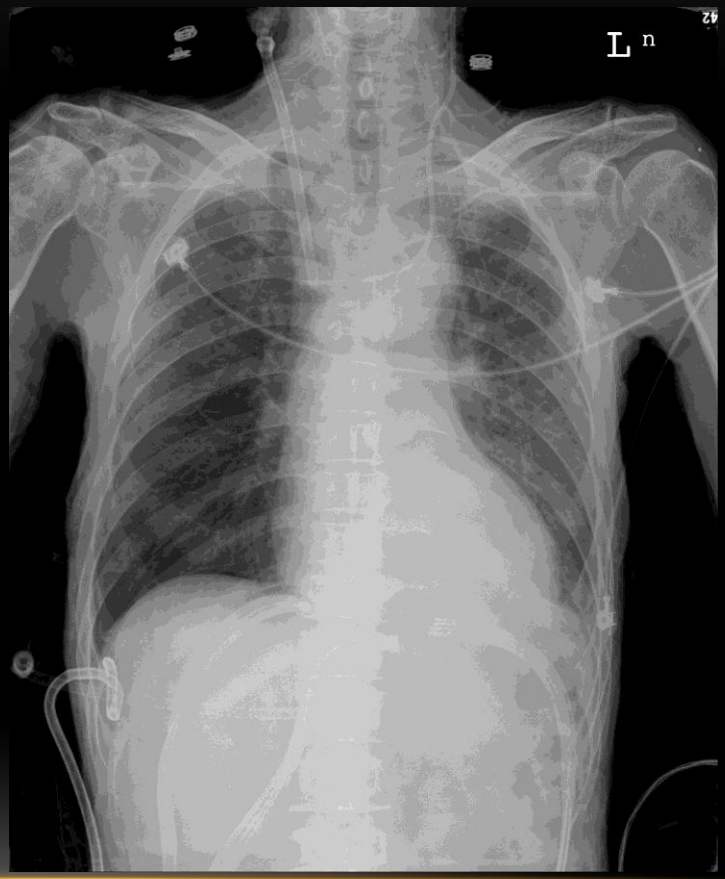
CASE PRESENTATION

BRIEF HISTORY

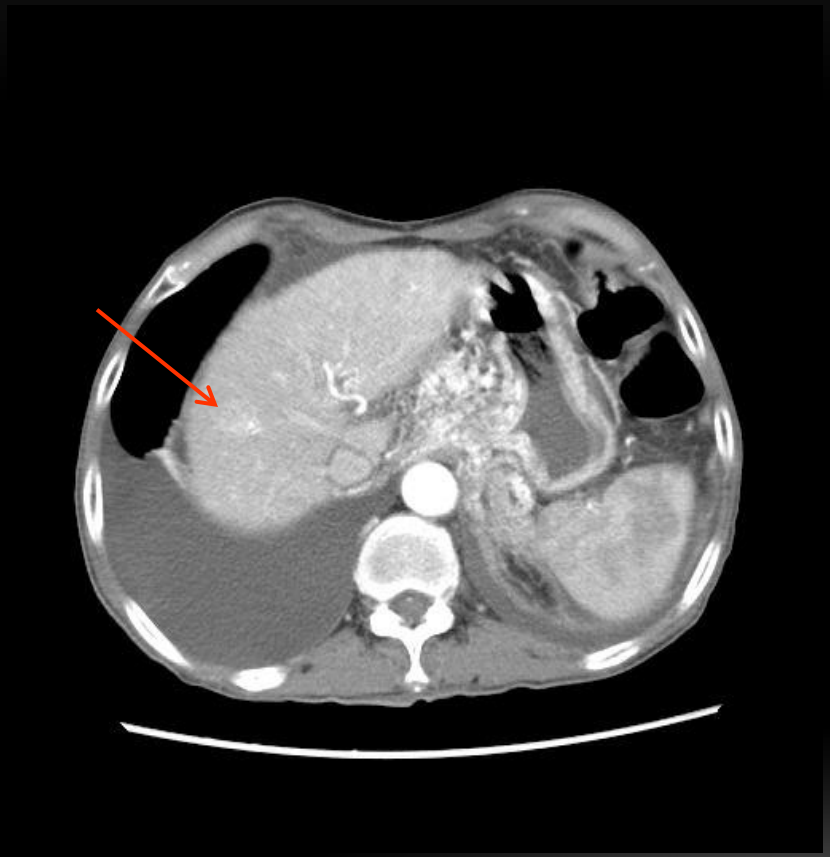
- 65-year-old man with a history of chronic hepatitis C with cirrhosis and type 2 DM
- Chief complaint: Progressive dyspnea for one week.
- Chest PA: Massive right pleural effusion and left pleural effusion
- CT: Cirrhosis and right hepatic nodule, 2 cm, S8
- Pig-tail insertion, right pleural cavity
- 12 days hospitalization: hepatic coma (442 umol/L)
- Referred for liver transplant

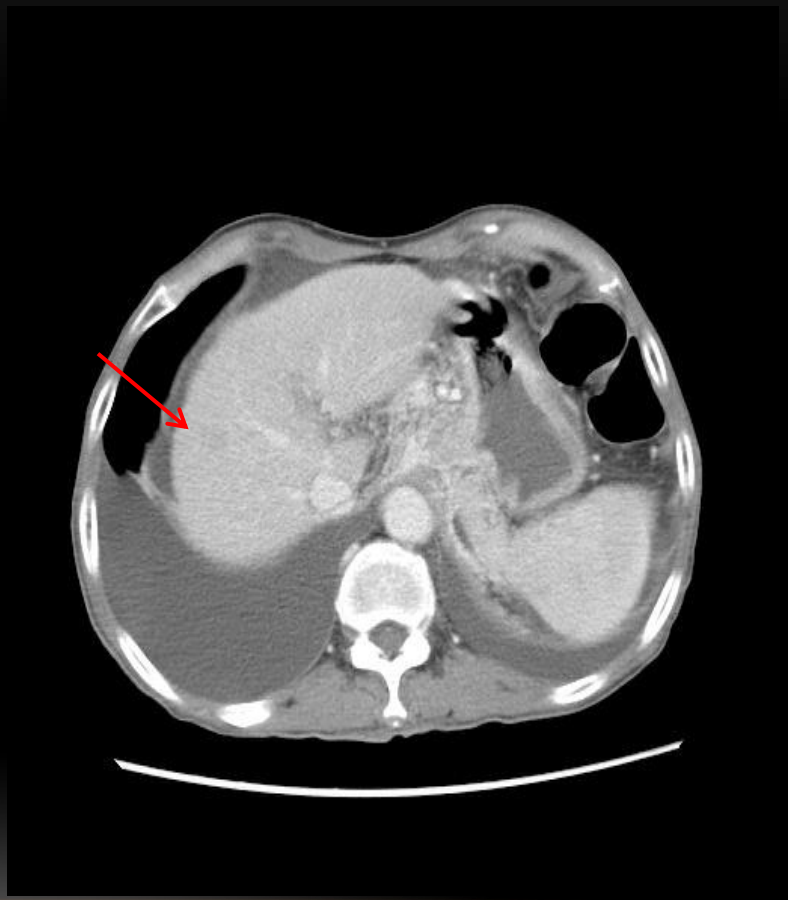
CHEST PA





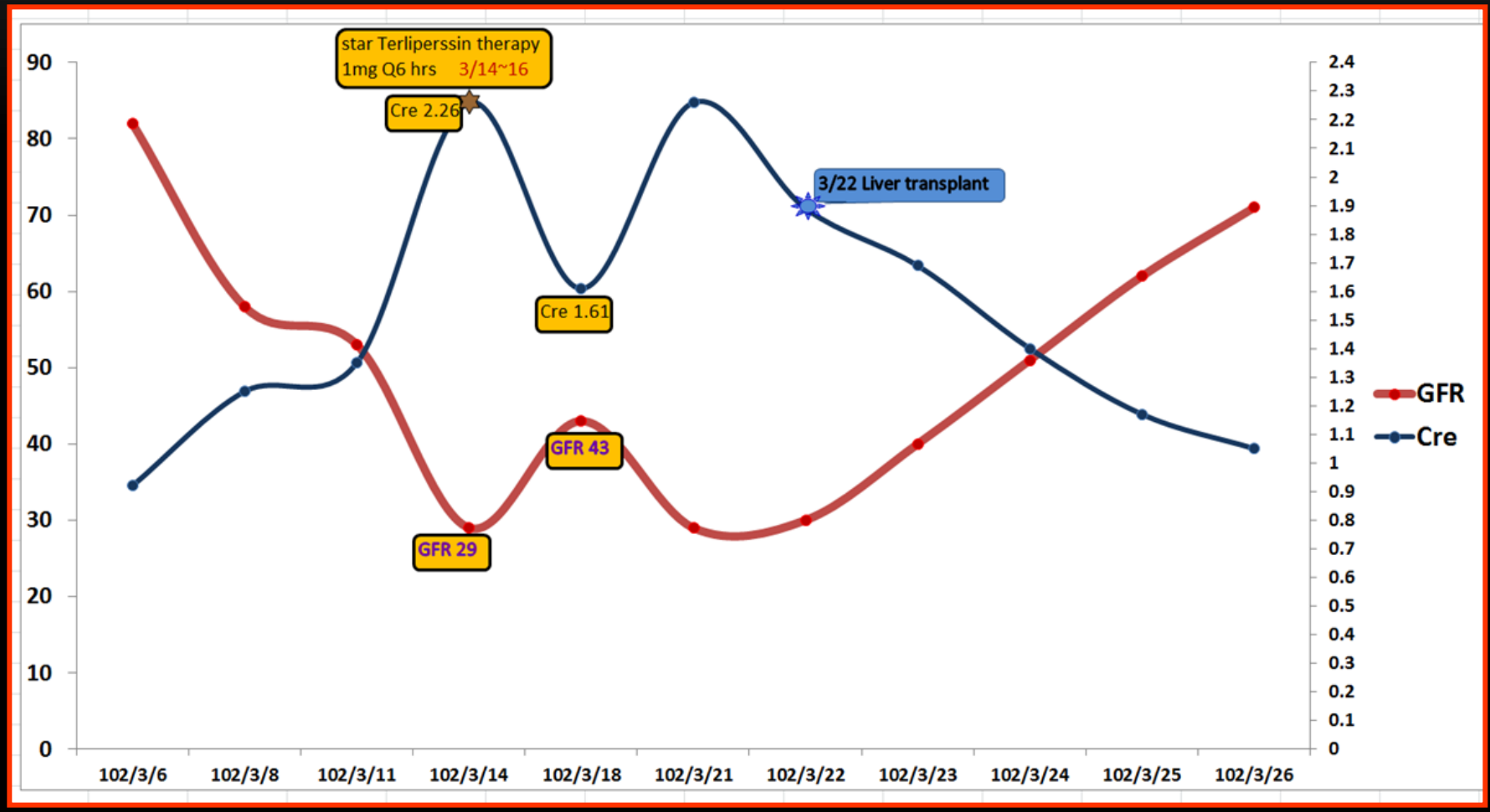
ABDOMINAL CT





LAB DATA

- CBC: Hb 12.2 g/dL, Platelet 72000/mm^3
- Biochemistry: Creatinine 0.92 mg/dL, total bilirubin 2.33 mg/dL , ALT 63 IU/L, AST 119 IU/L, albumin 2.7 g/dL.
- PT 25.2 sec, INR 2.32
- Child-Pugh Classification, Child C
- Model for End-Stage Liver Disease **(MELD score)** score 19 to 27


star Terliperssin therapy
1mg Q6 hrs 3/14~16
Cre 2.26
Cre 1.61
3/22 Liver transplant
GFR 43
GFR 29
90
80
70
60
50
40
30
20
10
0
2.4
2.3
2.2
2.1
2
1.9
1.8
1.7
1.6
1.5
1.4
1.3
1.2
1.1
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
GFR
Cre
102/3/6
102/3/8
102/3/11
102/3/14
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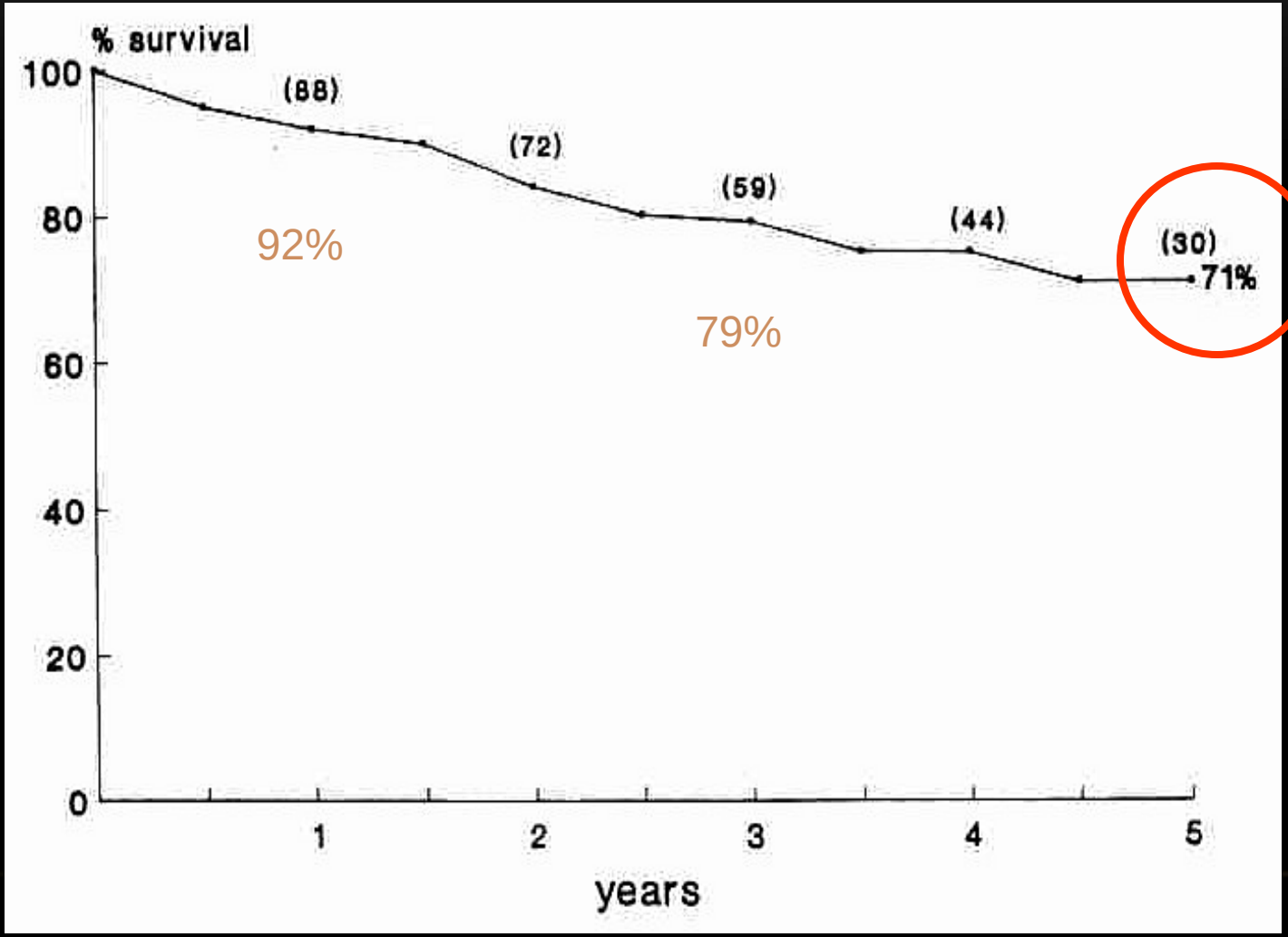
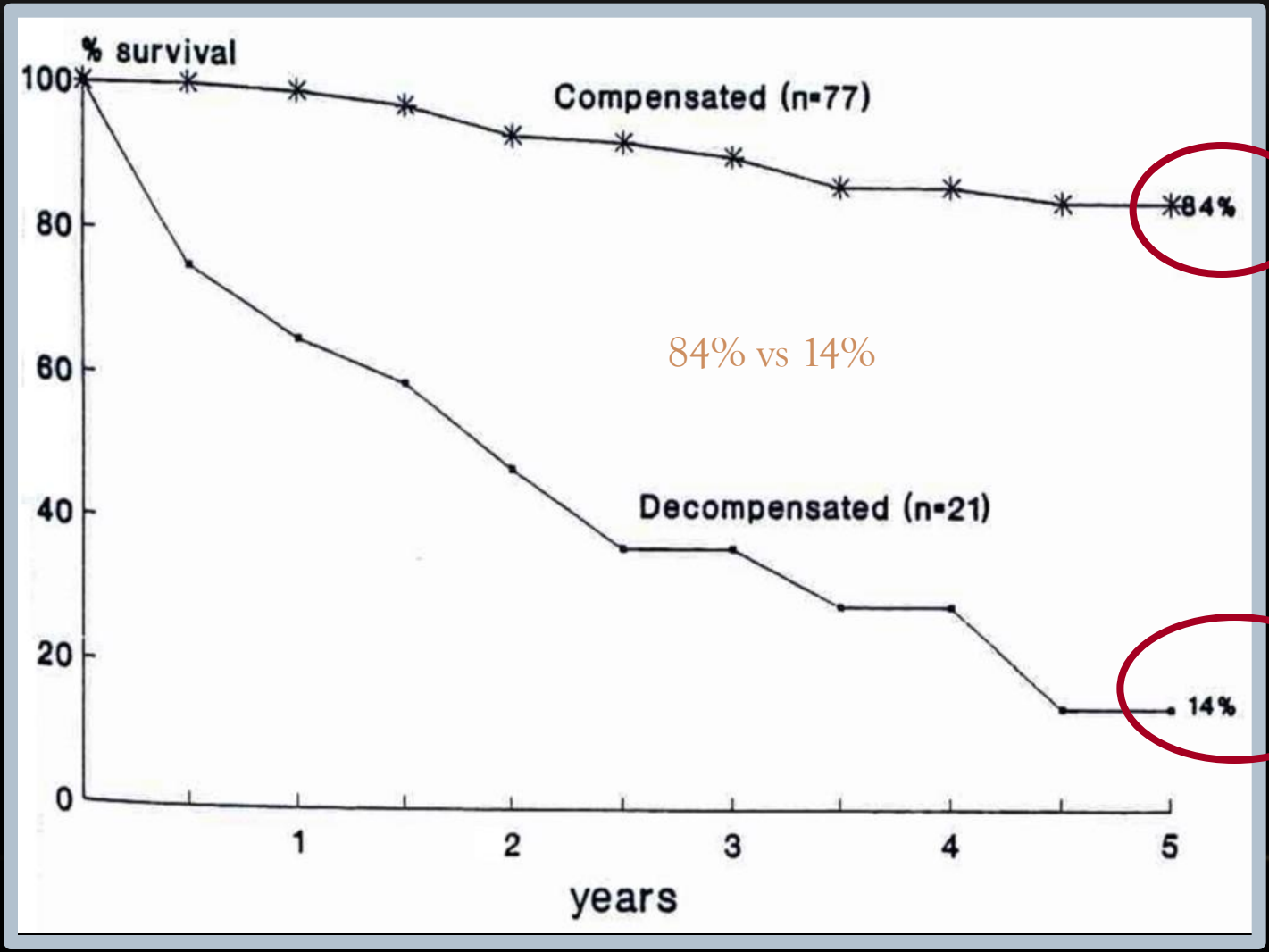
102/3/26

Survival of 98 patients with HBV related cirrhosis



De Jongh et al. Gastroenterology 1992

Survival and Compensation



De Jongh et al. Gastroenterology

Hepatorenal Syndrome: A Severe, but Treatable, Cause of Kidney Failure in Cirrhosis

Cláudia Fagundes, MD, and Pere Ginès, MD, PhD

Hepatorenal syndrome (HRS) is a unique type of kidney failure that occurs in advanced cirrhosis. It is characterized by functional impairment of the kidneys due to vasoconstriction of the renal arteries in the setting of preserved tubular function and absence of significant histologic abnormalities. Renal vasoconstriction in HRS is due to severe vasodilation of the splanchnic arteries associated with portal hypertension, leading to a decrease in effective arterial blood volume and arterial pressure. HRS commonly develops after a trigger, usually a bacterial infection, that disrupts the arterial circulation, but it also may occur spontaneously. There are 2 forms of HRS: type 1 is characterized by an acute progressive decrease in kidney function and very short survival without treatment, whereas type 2 features stable less severe kidney failure and longer survival compared with type 1. A liver transplant is the preferred treatment for HRS. Pharmacologic treatment with vasoconstrictors to reverse splanchnic vasodilation, together with albumin, is effective in 40%-50% of patients with type 1 HRS and improves survival. The drug of choice is the vasopressin analogue terlipressin. Renal replacement therapy should not be used as first-line therapy.

Am J Kidney Dis. 59(6):874-885.

Am J Kidney Dis. 59(6):874-885.

Diagnostic criteria of hepatorenal syndrome

Consensus conference, Chicago 1994

- Hepatic failure and portal hypertension
- Creatinine >1.5 mg/dl or GFR <40 mL/min
- No improvement after diuretic withdrawal and IV saline infusion (1500 ml)
- No shock, no ongoing bacterial infection, nephrotoxic agents or fluid losses
- Proteinuria <500 mg/dl, normal renal US

New proposal, San Francisco 2005

- Cirrhosis with ascites
- Creatinine >1.5 mg/dl
- No improvement after diuretic withdrawal and IV albumin infusion (1 g/kg.d) 48 h
- Absence of shock
- No recent nephrotoxic agents
- Proteinuria <500 mg/dl, microhematuria (<50 RBC/field), normal renal US

ALBUMIN AND TERLIPRESSIN DECREASE IN SERUM CREATININE LEVEL AND INCREASE IN ARTERIAL PRESSURE

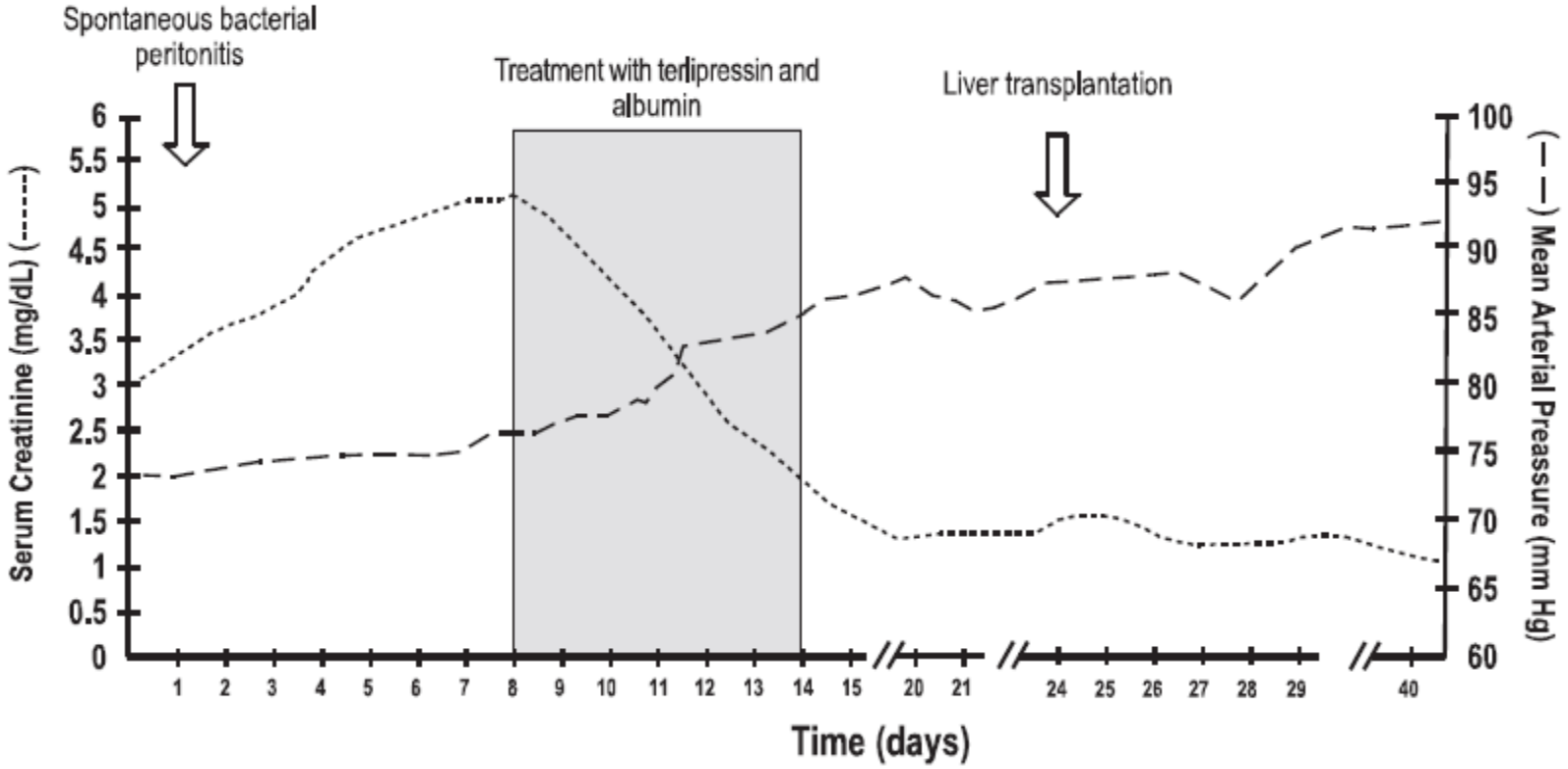


Figure 1. Serum creatinine concentration and mean arterial pressure in the patient described in the case vignette.

Am J Kidney Dis. 59(6):874-885.

PATHOGENESIS OF CIRCULATORY ABNORMALITIES IN COMPENSATED CIRRHOSIS AND HEPATORENAL SYNDROME

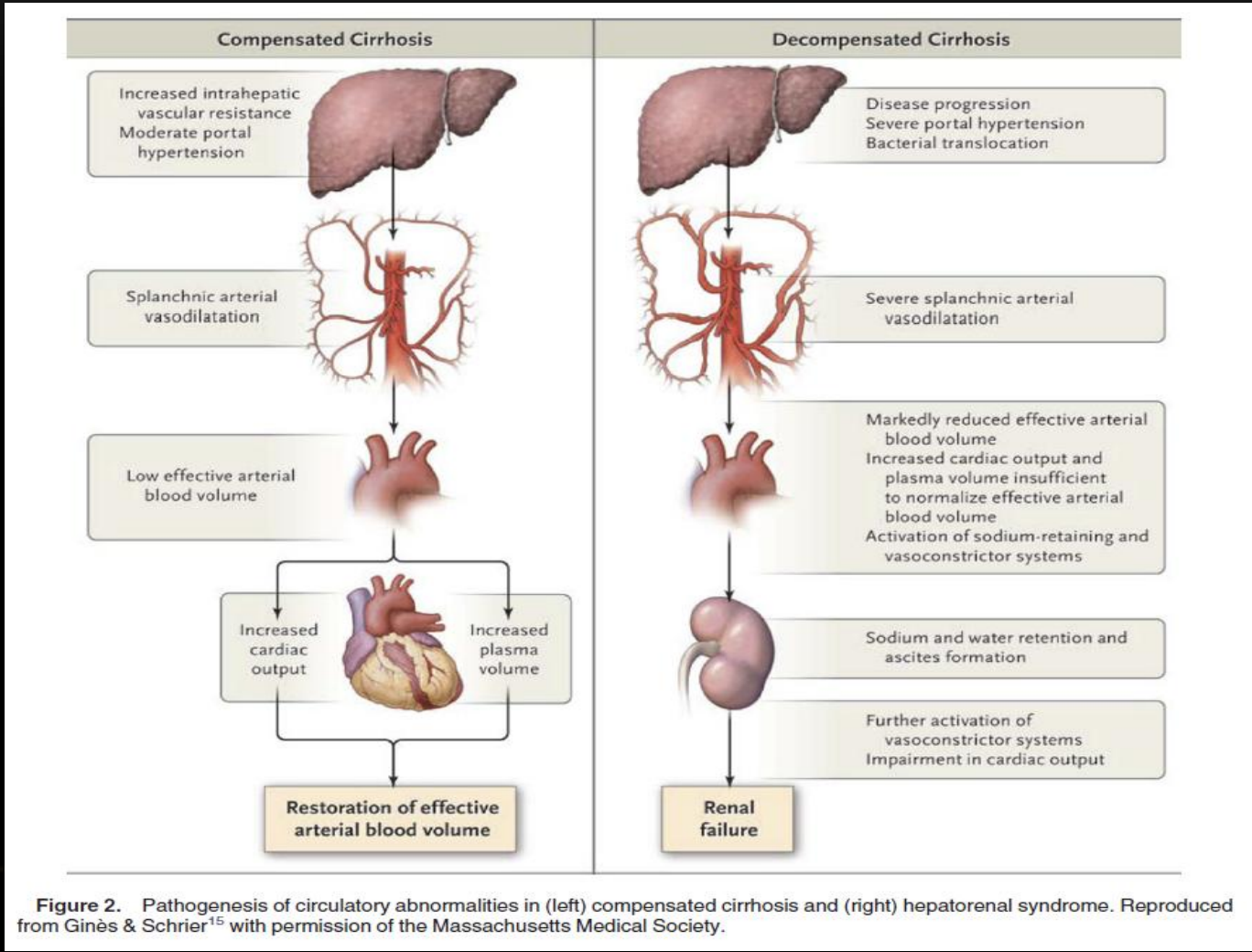


Figure 2. Pathogenesis of circulatory abnormalities in (left) compensated cirrhosis and (right) hepatorenal syndrome. Reproduced from Ginès & Schrier[15] with permission of the Massachusetts Medical Society.

Am J Kidney Dis. 59(6):874-885.

TYPE 1 HRS ARE THOSE OF ACUTE KIDNEY FAILURE WITH A RAPID INCREASE IN SERUM CREATININE LEVEL

Box 1. Clinical Types of HRS

Type 1: Rapidly progressive decrease in kidney function, defined as a 100% increase in serum creatinine to a final value >2.5 mg/dL (>221 μmol/L) in <2 weeks. The clinical presentation is usually that of acute kidney failure. Average median survival is only 2 weeks if not treated.

Type 2: Stable or slowly progressive decrease in kidney function that does not meet the criteria of type 1. The clinical picture is that of ascites refractory to diuretic therapy. Average median survival is ~6 months.

Abbreviation: HRS, hepatorenal syndrome.

Source: Salerno et al.[5]

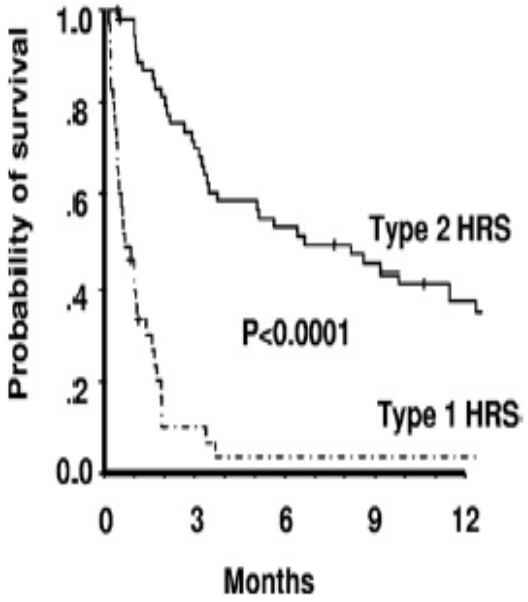


Figure 3. Survival of patients with cirrhosis according to type of hepatorenal syndrome (HRS). Reproduced from Alessandria et al[46] with permission of John Wiley & Sons.

Am J Kidney Dis. 59(6):874-885.

MODEL FOR END-STAGE LIVER DISEASE (MELD SCORE) IS A PREDICT MORTALITY WITHIN THREE MONTHS OF A LIVER TRANSPLANT

- MELD uses serum bilirubin, serum creatinine, and the international normalized ratio for prothrombin time (INR) to predict survival
- MELD = 3.78[Ln serum bilirubin (mg/dL)] + 11.2[Ln INR] + 9.57[Ln serum creatinine (mg/dL)] + 6.43

PATIENTS WHO DIED HAD SIGNIFICANTLY HIGHER SERUM CREATININE , INR SCORES AND SERUM BILIRUBIN

Table 3. Meld Parameters at Time of Listing in Patients Who Survived and Patients Who Died While on the Waiting List

	Survival (n = 1859)	Transplanted (n = 1040)	Died within 3 months (n = 412)
Creatinine	1.2 ± 1.4 (1.0)	1.4 ± 1.2 (1.1)[a]	2.0 ± 1.6 (1.4)[a]
Bilirubin	4.2 ± 3.5 (3.0)	8.0 ± 9.4 (4.0)[a]	12.2 ± 11.2 (7.6)[a]
INR	1.6 ± 0.5 (1.5)	1.9 ± 0.8 (1.7)[a]	2.2 ± 1.0 (1.9)[a]
MELD	16.9 ± 5.4 (16.3)	21.5 ± 8.3 (19.9)[a]	27.0 ± 9.6 (25.5)[a]
CTP	10.5 ± 1.4 (10.0)	11.2 ± 1.9 (11.0)	12.1 ± 1.6 (12.0)

Wiesner et al. Gastroenterology 2003;124 :91-96

MORTALITY INCREASED IN PROPORTION TO THE INCREASE IN THE MELD SCORE

Table 4. Three-Month Mortality Based on Meld and CTP Score

	MELD					CTP		
	<9	10–19	20–29	30–39	>40	<7–9	10–12	13–15
No.	124	1800	1098	295	120	318	2357	588
Mortality	1.9	6.0	19.6	52.6	71.3	4.3	11.2	40.1
Mortality + too sick	2.9	7.7	23.5	60.2	79.3	5.6	13.4	48.5

NOTE. There were 66 patients for whom the CTP score was not available, and 108 patients had a CTP score of <7 and were granted 2B status because of HCC or metabolic liver disease and were not included in this analysis.

Wiesner et al. Gastroenterology 2003;124 :91-96

MELD SCORE AND THE CTP SCORE, BOTH SCORES WERE NOTED TO VARY CONSIDERABLY AT EACH SEVERITY SCORE

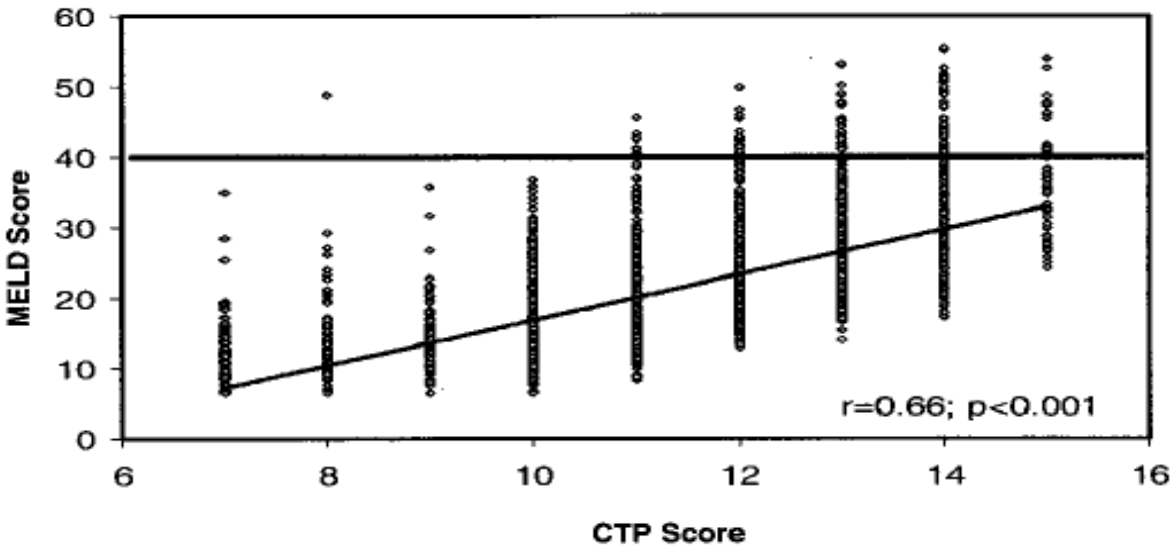


Figure 1. The relationship between the MELD score and CTP at time of listing on the OPTN waiting list. Patients with hepatocellular cancer or metabolic liver disease with a Child-Pugh score of less than 7 were excluded in the analysis.

Wiesner et al. Gastroenterology 2003;124 :91-96

THE RELATIONSHIP BETWEEN THE MELD SCORE AND ESTIMATED 3-MONTH MORTALITY IN PATIENTS WITH CHRONIC LIVER DISEASE

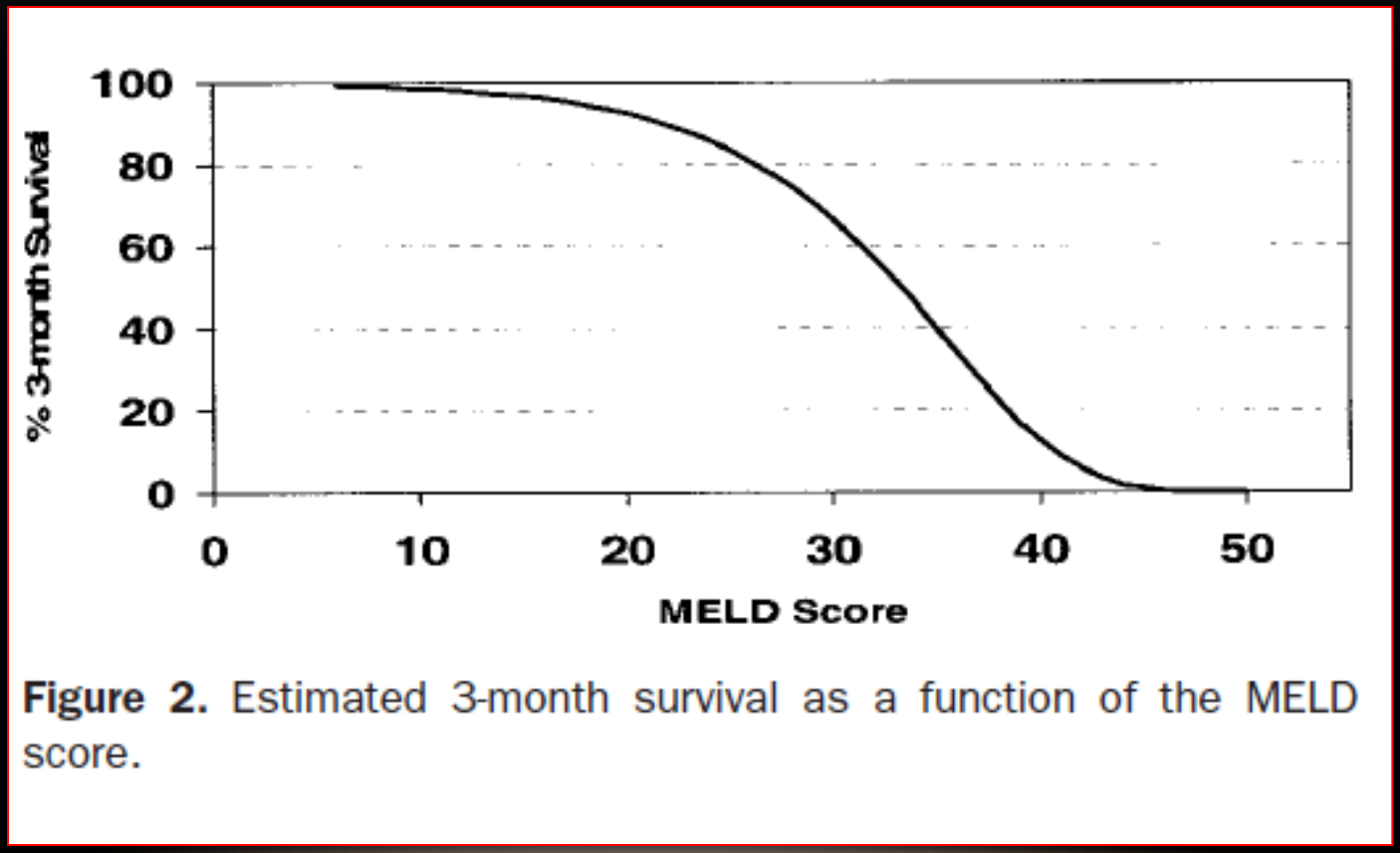


Figure 2. Estimated 3-month survival as a function of the MELD score.

Wiesner et al. Gastroenterology 2003;124 :91-96

MELD SCORE IS SUPERIOR TO THE CTP SCORE IN RANKING PATIENTS ACCORDING TO SEVERITY OF THEIR LIVER DISEASE AND RISK OF DYING

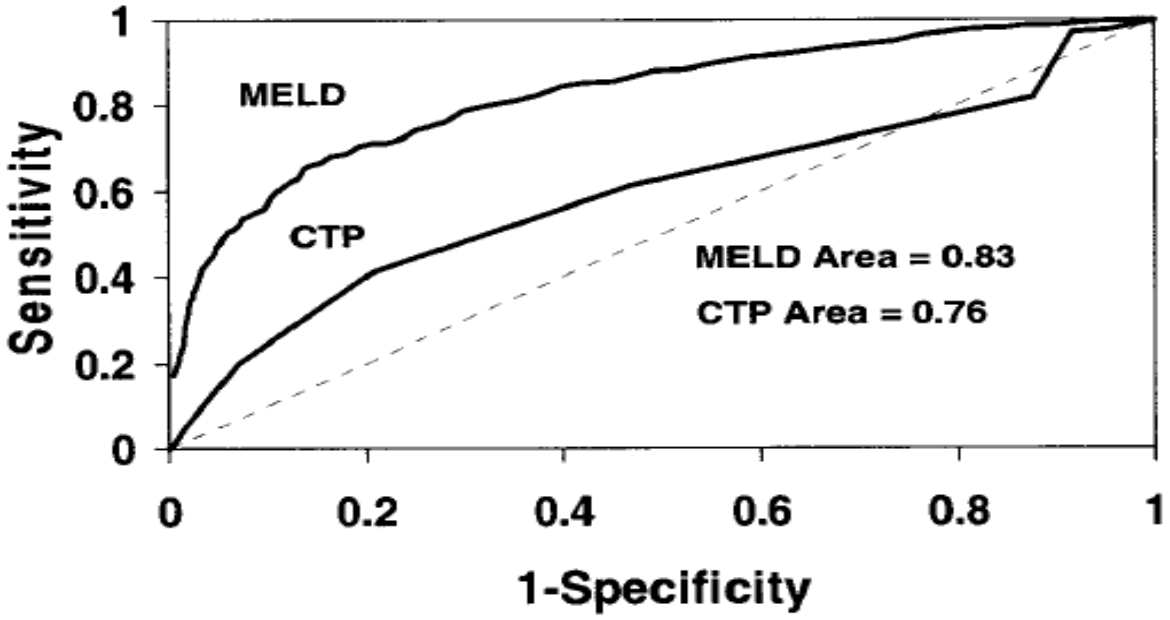


Figure 3. The area under the receiving operating curve for the MELD score and CTP score with 3-month mortality as the end point. The difference was significant ($P < 0.001$). *Dotted line* represents the ROC based on chance alone and has a c-statistic of 0.5.

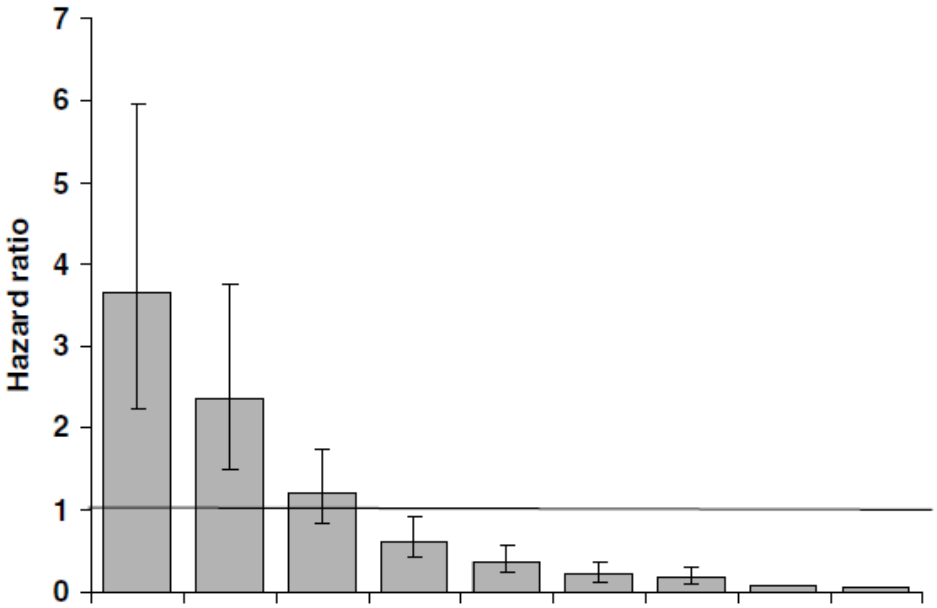
Wiesner et al. Gastroenterology 2003;124 :91-96

DETERMINING THE NEED FOR LIVER TRANSPLANTATION

- Patients with cirrhosis should be referred for transplantation when they develop evidence of hepatic dysfunction (CTP > 7 and MELD > 10)
- When they experience their first major complication (ascites, variceal bleeding, or hepatic encephalopathy)

Karen F et al. Hepatology 2005

PATIENTS WITH MELD SCORES LESS THAN 14, MORTALITY WITH TRANSPLANTATION HIGHER THAN THAT OF PATIENTS WITH THE SAME MELD SCORE NOT TRANSPLANTED



MELD	6-11	12-14	15-17	18-20	21-23	24-26	27-29	30-39	≥40
Hazard Ratio	3.64	2.35	1.21	0.62	0.38	0.22	0.18	0.07	0.04
p-values	<0.001	<0.001	0.41	<0.01	<0.001	<0.001	<0.001	<0.001	<0.001

Figure 3: Comparison of mortality risk expressed as hazard ratio by MELD score for recipients of liver transplants compared to candidates on the liver transplant waiting list.

Merion RM et al. Am J Transplant 2005;5:307-313

LIVER TRANSPLANT IN RENAL FAILURE

- The presence of renal insufficiency is an important predictor of postoperative renal failure and mortality after liver transplantation, and hence a thorough pretransplantation evaluation of renal function is important
- Rapidly progressive hepatorenal syndrome (type 1) has an ominous prognosis and usually is reversed by transplantation, patients with this condition should have an expedited referral for evaluation
- Selected patients with chronic renal and liver disease should be considered for combined liver–kidney transplantation

Karen F et al. Hepatology 2005

DEFINITION OF RESPONSE TO TERLIPRESSIN AND ALBUMIN

- Complete response: Reduction of serum creatinine below 133 umol/L (0.92 mg/dL)at the end of treatment
- Partial response: Reduction in serum creatinine greater than 50% of the pre-treatment value but with an end-of-treatment value equal to or greater than 133 umol/L

Gastroenterology 2008;134:1352–1359.

EFFECTS OF TREATMENT OF HEPATORENAL SYNDROME BEFORE TRANSPLANTATION ON POSTTRANSPLANTATION OUTCOME. A CASE-CONTROL STUDY

Tea Restuccia[1,2,3], Rolando Ortega[1,2,3], Monica Guevara[1,2,3], Pere Ginès[1,2,3,*], Carlo Alessandria[1,2,3], Osman Ozdogan[1,2,3], Miquel Navasa[1,2], Antoni Rimola[1,2], Juan Carlos Garcia-Valdecasas[4], Vicente Arroyo[1,2,3], Juan Rodés[1,2,3]

[1]*Liver Unit, Institute for Digestive Diseases, Hospital Clínic, University of Barcelona, Villarroel 170, Barcelona 08036, Catalunya, Spain*
[2]*Institut d'Investigacions Biomèdiques August Pi-Sunyer (IDIBAPS), Barcelona, Catalunya, Spain*
[3]*Instituto Reina Sofía de Investigación Nefrológica, Barcelona, Spain*
[4]*Department of Surgery, Institute for Digestive Diseases Hospital Clinic, Barcelona, Catalunya, Spain*

Restuccia T et al. J Hepatol 2004;40:140–146

PATIENTS

Table 1
Response to treatment and transplantation in the 21 transplant candidates with hepatorenal syndrome (HRS) treated with vasopressin analogues divided according to the type of hepatorenal syndrome

	Transplanted	Non-transplanted
Type 1 HRS ($n = 11$)		
Responders ($n = 6$)	3/6	3/6
Non-responders ($n = 5$)	1/5	4/5
Type 2 HRS ($n = 10$)		
Responders ($n = 10$)	6/10	4/10
Non-responders ($n = 0$)	–	–

follow-up. Fig. 1 shows the transplant-free survival of the 21 patients with HRS treated divided in two groups according to response to therapy. Median transplant-free survival was 5 months in responders as compared with only 0.4 months in non-responders ($P < 0.001$).

Restuccia T et al. J Hepatol 2004;40:140–146

MEDIAN TRANSPLANT-FREE SURVIVAL WAS 5 MONTHS IN RESPONDERS AS COMPARED WITH ONLY 0.4 MONTHS IN NON-RESPONDERS

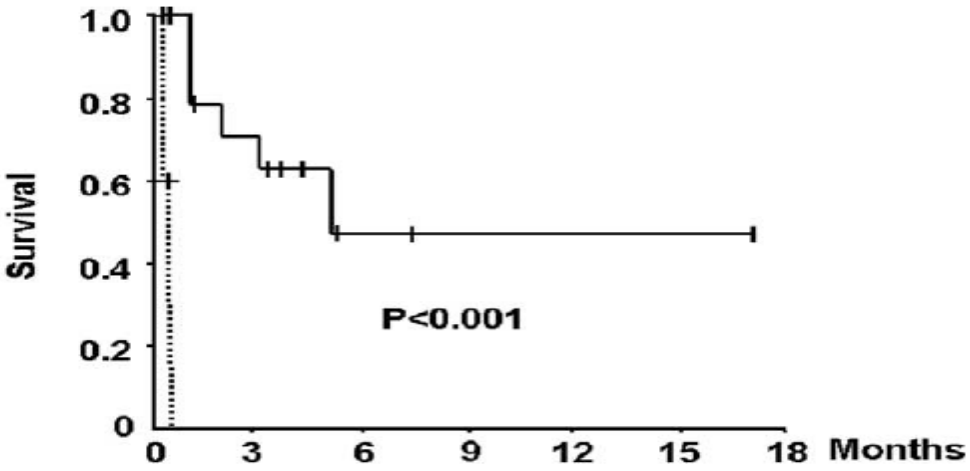


Fig. 1. Probability of transplant-free survival in the 21 patients candidates to transplantation who received treatment with vasopressin analogues for Hepatorenal syndrome divided according to response to therapy: responders (continuous line) and non-responders (discontinuous line). The small vertical lines in each curve represent the time of transplantation of the patients who were transplanted during follow-up.

Restuccia T et al. J Hepatol 2004;40:140–146

NO DIFFERENCES IN RENAL FUNCTION PARAMETERS BETWEEN HRS-TREATED AND NO-HRS

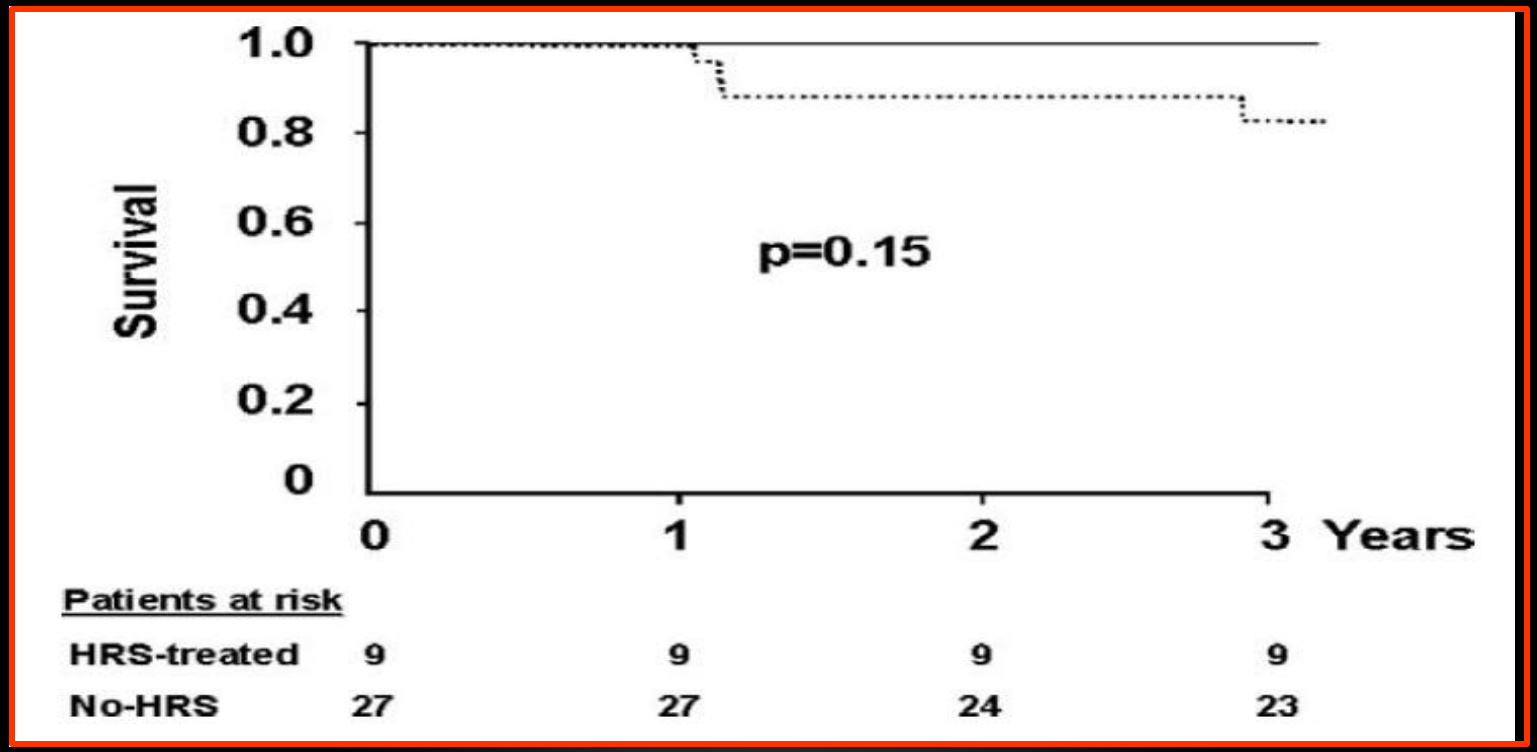
Table 2
Baseline characteristics of patients with cirrhosis and hepatorenal syndrome treated (HRS-treated), both immediately before treatment and at transplantation, and of patients with cirrhosis without HRS (no-HRS) at transplantation

	HRS-treated ($n = 9$)		No-HRS ($n = 27$)	P[a]
	Before treatment	At transplantation	At transplantation	
Age (years)	50 ± 2	50 ± 2	52 ± 1	0.4
Sex (male/female)	4/5	4/5	18/9	0.4
Etiology of cirrhosis				0.4
Alcoholic	2	2	7	
HCV-positive	4	4	13	
HBV-positive	2	2	1	
Other	1	1	6	
Bilirubin (mg/dL)	13 ± 6	16 ± 7	5 ± 1	0.8
Albumin (g/l)	33 ± 3	35 ± 1	29 ± 1	0.003
Prothrombin time (%)	41 ± 7	46 ± 10	48 ± 4	0.6
Child-Pugh				
Class B/C	2/7	4/5	12/15	1
Score	11 ± 0.7	10 ± 0.7	10 ± 0.3	0.8
Ascites (0/1/2/3)	0/0/3/6	0/1/4/4	3/5/13/6	0.4
Serum creatinine (mg/dL)	2.7 ± 0.4	1.3 ± 0.2	0.9 ± 0.04	0.06
Blood urea nitrogen (mg/dL)	66 ± 8	52 ± 10	20 ± 3	0.001
Serum sodium (mEq/l)	127 ± 2	134 ± 1	132 ± 1	0.4

[a] P between HRS-treated patients and no-HRS patients at the time of transplantation.

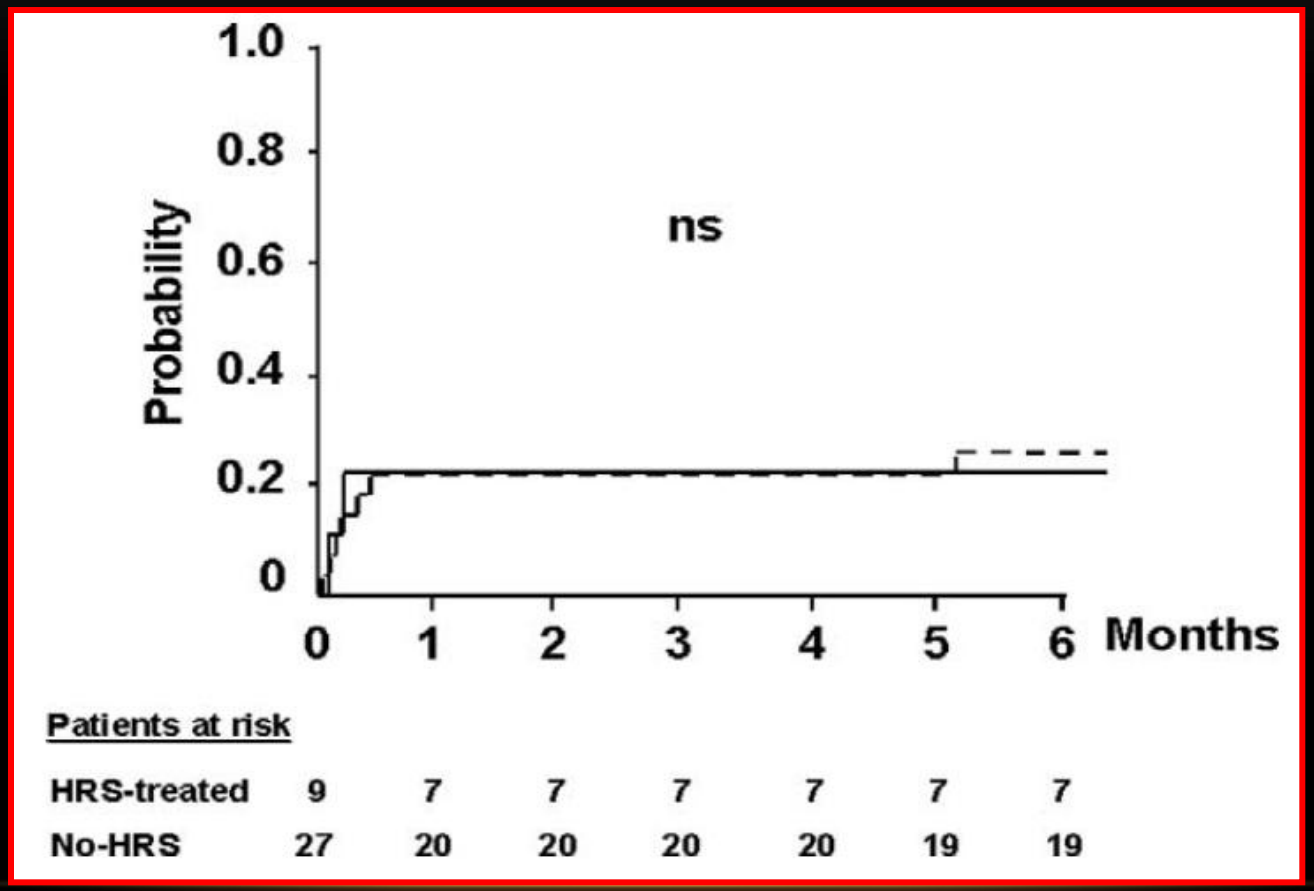
Restuccia T et al. J Hepatol 2004;40:140–146

THREE-YEAR PROBABILITY OF SURVIVAL WAS 100% IN THE HRS-TREATED GROUP AND 83% IN THE CONTROL GROUP



Restuccia T et al. J Hepatol 2004;40:140–146

THE PROBABILITY OF DEVELOPING RENAL FAILURE WAS SIMILAR IN THE TWO GROUPS



Restuccia T et al. J Hepatol 2004;40:140–146

NO SIGNIFICANT DIFFERENCES WERE OBSERVED IN BUN AND Cr BETWEEN THE TWO GROUPS

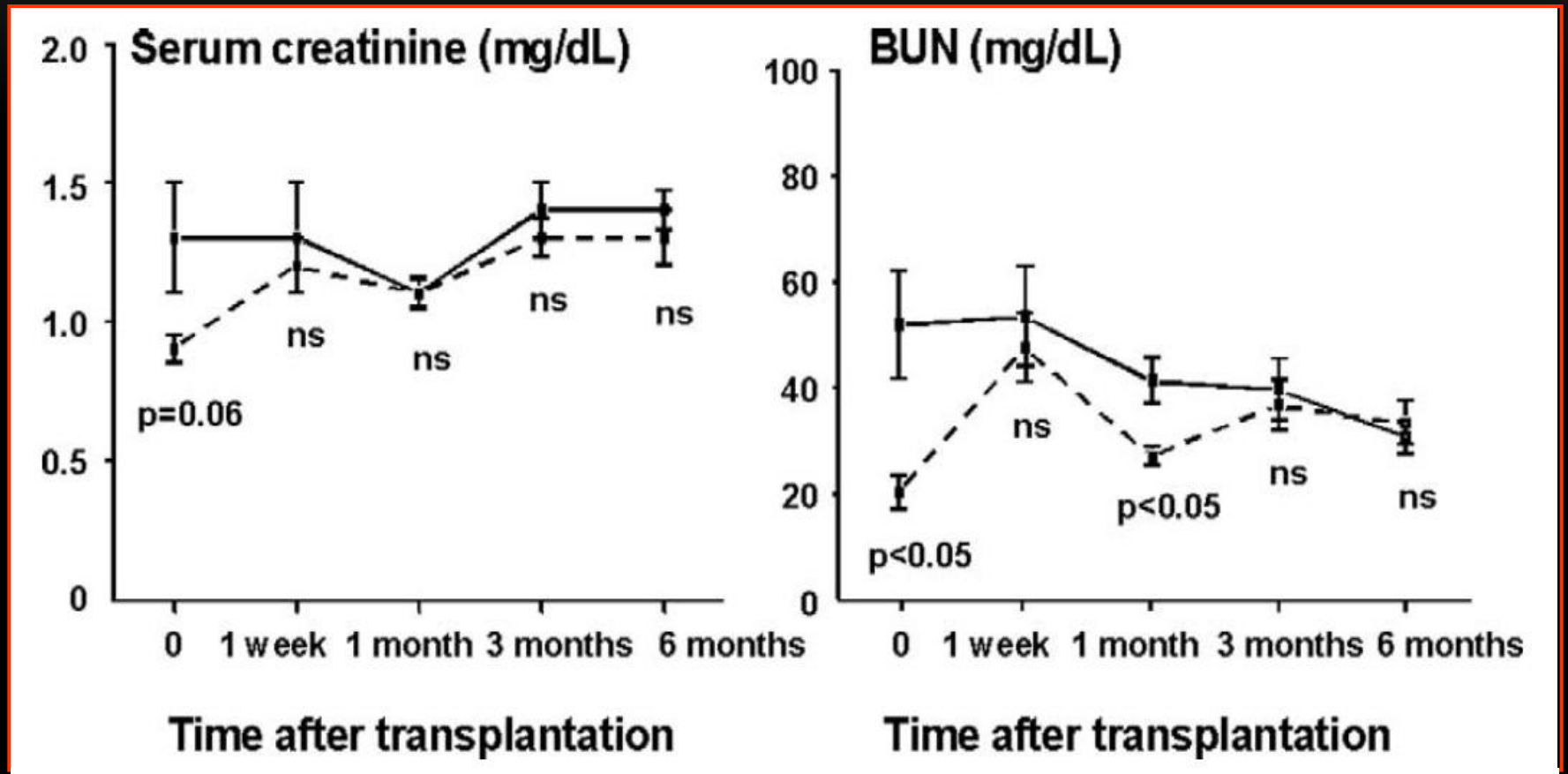


Fig. 3. Mean values of serum creatinine and BUN during the first 6 months after transplantation in patients with hepatorenal syndrome treated with vasopressin analogues before transplantation (continuous line) and patients without renal failure (discontinuous line).

Restuccia T et al. J Hepatol 2004;40:140–146

IMPACT OF LIVER TRANSPLANTATION ON THE SURVIVAL OF PATIENTS TREATED FOR HEPATORENAL SYNDROME TYPE 1

Thomas D. Boyer,[1] Arun J. Sanyal,[2] Guadalupe Garcia-Tsao,[3,4] Frederick Regenstein,[5] Lorenzo Rossaro,[6] Beate Appenrodt,[7] Veit Gülberg,[8,9] Samuel Sigal,[10] Alice S. Bexon,[11] Peter Teuber[11] and the Terlipressin Study Group*

[1]Liver Research Institute, Department of Internal Medicine, University of Arizona College of Medicine, Tucson, AZ; [2]Division of Gastroenterology, Hepatology, and Nutrition, Department of Internal Medicine, Virginia Commonwealth University, Richmond, VA; [3]Digestive Diseases Section, Department of Internal Medicine, Yale University, New Haven, CT; [4]VA Connecticut Healthcare System, New Haven, CT; [5]Division of Gastroenterology, Department of Internal Medicine, Tulane University Health Sciences Center, New Orleans, LA; [6]Division of Gastroenterology and Hepatology, Department of Internal Medicine, University of California Davis Medical Center, Sacramento, CA; [7]Department of Medicine I, University of Bonn, Bonn, Germany; [8]Department of Medicine II, Klinikum Grosshadern, and [9]Liver Center Munich, Division of Gastroenterology, Department of Medicine, Ludwig-Maximilians University of Munich, Munich, Germany; [10]New York University Medical Center, New York, NY; and [11]Orphan Therapeutics, LLC, Lebanon, NJ

LIVER TRANSPLANTATION 2011:17:1328-1332

IMPACTION OF HEPATORENAL SYNDROME ON LIVER TRANSPLANT

Liver transplantation is considered the treatment of choice for patients with cirrhosis and HRS because it ‘‘allows for both the liver disease and associated renal failure to be cured’’.

PATIENTS

TABLE 1. Patients Who Were Eligible for Liver Transplantation (n = 99)

Parameter	Terlipressin Group (n = 47)	Placebo Group (n = 52)
Transplant patients [n (%)]	18 (38)	17 (33)
Time to transplantation (days)*	31 (1-142)	21 (5-113)
Drug exposure: doses (n)	23	19
Baseline serum creatinine level (mg/dL)	3.1	3.5
Last on-treatment serum creatinine level (mg/dL)	2.8	3.8
Dialysis before transplantation (%)	39	53
Baseline serum sodium level (mmol/L)	130	133
Last on-treatment sodium level (mmol/L)	134	135
Baseline MELD score	33	32
Last on-treatment MELD score	31	32
Living on day 180 (%)		
Transplant patients	100	94
Nontransplant patients	34	17

*The data are presented as means and ranges.

Liver transpl 2011:17:1328-1332

SURVIVAL RATE WAS SLIGHTLY BETTER FOR THOSE RECEIVING TERLIPRESSIN (34% VERSUS 17%) IN NONTRANSPLANT PATIENTS

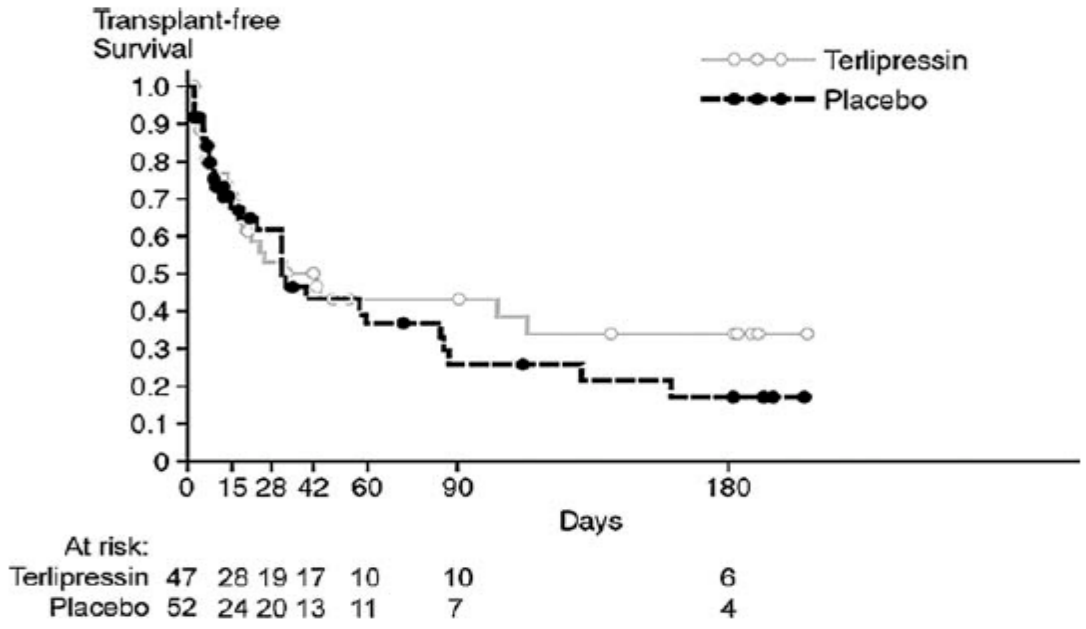


Figure 1. Transplant-free survival in patients receiving terlipressin plus albumin (n = 47) and patients receiving albumin alone (n = 52). The 99 patients came from 32 centers offering liver transplantation ($P = 0.40$).

Liver tanspll 2011:17:1328-1332

THE SURVIVAL AT WAS EXCELLENT FOR THOSE WHO UNDERWENT TRANSPLANTATION

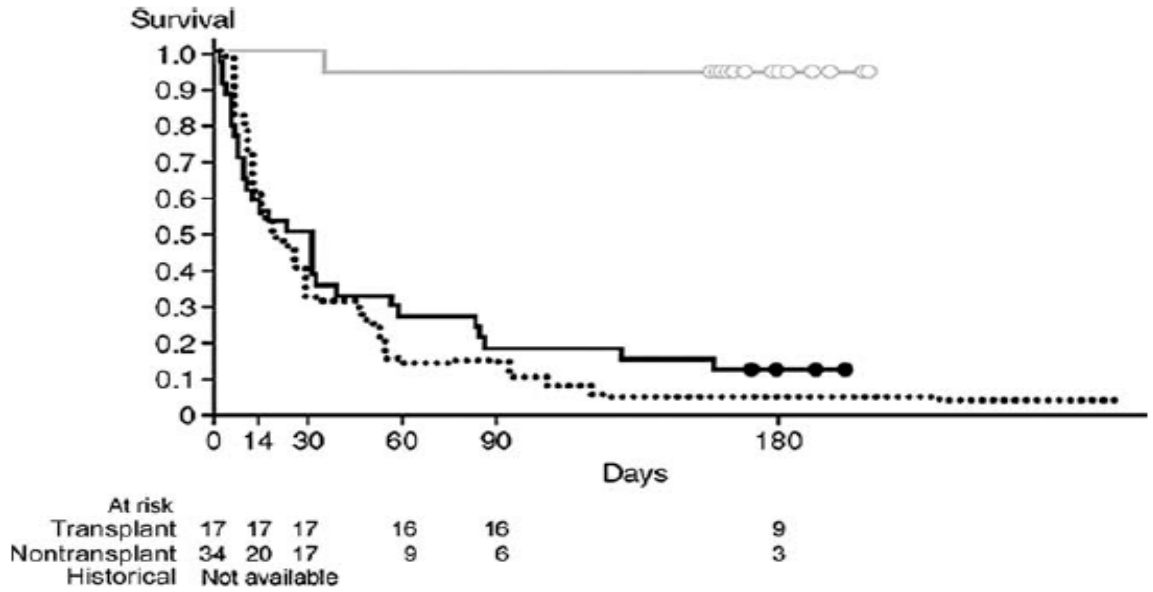


Figure 2. Cross-trial comparison of the overall survival of (-○-) albumin-only (placebo) patients who underwent liver transplantation (n = 17), (-●-) albumin-only (placebo) patients who did not undergo liver transplantation (n = 34), and (···) a historical cohort of untreated, nontransplant patients with cirrhosis after a diagnosis of HRS1. The historical control group was taken from Ginès et al.[15]

Liver tanspl 2011:17:1328-1332

SURVIVAL WAS SIGNIFICANTLY BETTER FOR THE HRS RESPONDERS VERSUS THE NONRESPONDERS

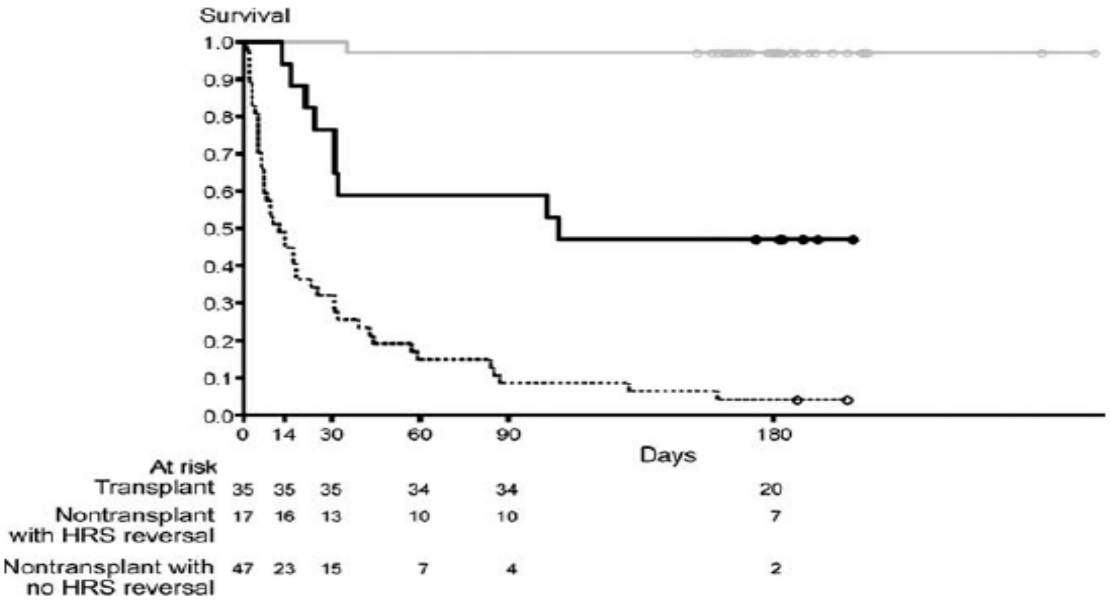


Figure 3. Overall survival of (-○-) transplant patients (n = 35), (-●-) nontransplant patients who achieved HRS reversal (n = 17), and (--○--) nontransplant patients who did not achieve HRS reversal (n = 47). The difference between the transplant group and the 2 nontransplant groups was significant ($P < 0.001$). The difference between those achieving HRS reversal and those not achieving HRS reversal was also significant ($P < 0.001$).

Liver transpl 2011:17:1328-1332

HEPATORENAL SYNDROME, MELD SCORE AND LIVER TRANSPLANTATION: AN EVOLVING ISSUE WITH RELEVANT IMPLICATIONS FOR CLINICAL PRACTICE

Paolo Angeli, Pere Gines

Paolo Angeli[1,*], Pere Gines[2,3]

[1]*Department of Medicine, Unit of Hepatic Emergencies and Liver Transplantation, University of Padova, Italy;* [2]*Liver Unit, Hospital Clínic, University of Barcelona School of Medicine, Villarroel 170, 08036 Barcelona, Catalunya, Spain;* [3]*Institut d'Investigacions Biomèdiques August Pi-Sunyer (IDIBAPS), Centro de Investigación Biomédica en Red de Enfermedades Hepáticas y Digestivas (CIBEREHED), Spain*

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J Hepatol 2012:7 :1135–1140

LT ALONE OR COMBINED LIVER–KIDNEY TRANSPLANTATION (CLKT) IN NON-RESPONDERS TO TERLIPRESSIN AND ALBUMIN

Frontiers in Liver Transplantation

Table 1. UNOS recommendations for combined liver-kidney transplantation (CLKT) in 2006 [36] and 2007 [37].

UNOS recommendations in 2006	UNOS recommendations in 2007
Patients with CKD a measured CrCl [or preferentially an iothalamate clearance] of ≤30 ml/min	Patients with ESRD
Patients with AKI and/or HRS on dialysis for ≥6 wk. CLKT was not recommended in patients with AKI not requiring dialysis	Patients with CKD with GFR ≤30 ml/min
Patients with prolonged AKI with kidney biopsy showing fixed renal damage	Patients with AKI including HRS with creatinine ≥2 mg/dl and dialysis ≥8 wk
	Patients with evidence of CKD and kidney biopsy demonstrating >30% glomerulosclerosis or 30% fibrosis

CKD, chronic kidney disease; CrCl, creatinine clearance; ESRD, End Stage Renal disease; AKI, acute kidney injury; HRS, hepatorenal syndrome.

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LT ALONE OR COMBINED LIVER–KIDNEY TRANSPLANTATION (CLKT) IN NON-RESPONDERS TO TERLIPRESSIN AND ALBUMIN

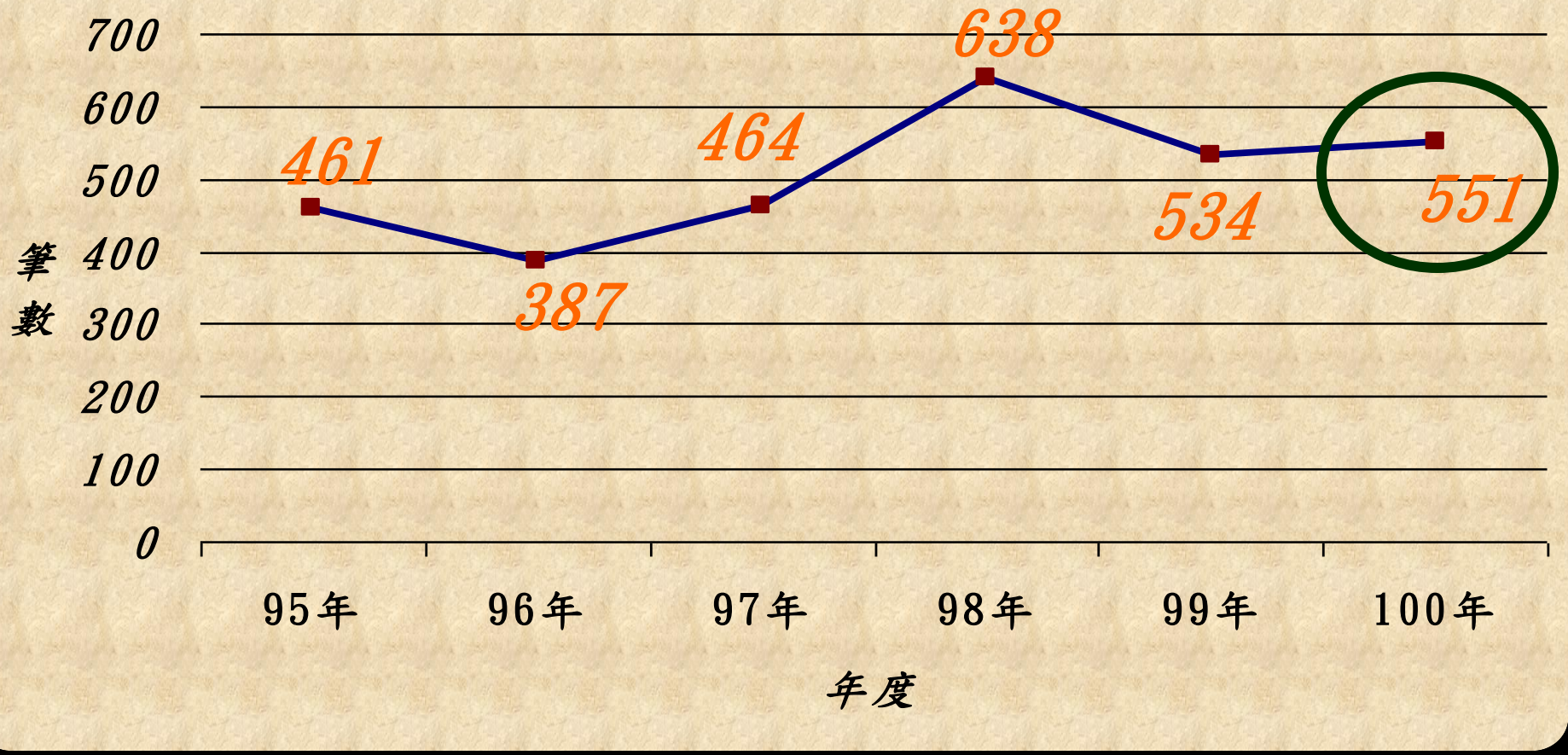
Table 2. Current UNOS recommendations for combined liver–kidney transplantation (CLKT) [38].

a) CKD requiring dialysis
b) CKD not requiring dialysis: documentation of both GFR ≤30 ml/min [by MDRD6 or iothalamate measurement] and proteinuria [>3 g protein per day with 24 h protein measurement or urine protein/creatinine ratio >3] is required
c) Sustained AKI requiring dialysis: documentation of dialysis for 6 wk or more [defined as dialysis at least twice a week for 6 consecutive weeks] is required
d) Sustained AKI not requiring dialysis: documentation of a GFR ≤25 ml/min for 6 wk or more by MDRD6 or direct measurement [iothalamate or iohexol] is required at least once a week
e) Sustained AKI: patients may also qualify for CLKT listing with a combination of time in categories (c) and (d) above for a total of 6 wk
f) Metabolic disease

CKD, chronic kidney disease; GFR, glomerular filtration rate; MDRD6, modification of diet in renal disease formula 6; AKI, acute kidney injury.

J Hepatol 2012:7 :1135–1140

民國95年~100年肝細胞癌新診斷人數



95-96年肝細胞癌治療分析表

	治癒性			姑息性
	OP	RFA	PEI	TACE
總計	223	30	88	231
95年stage1	56	5	23	22
95年stage2	29	5	21	29
95年stage3	24	1	13	52
95年stage4	1	0	0	22
96年stage1	67	12	19	19
96年stage2	20	7	12	31
96年stage3	26	0	0	47
96年stage4	0	0	0	9

97-99年肝細胞癌治療分析表

	根除性				支持性
	liver transplant	OP	RFA	PEI	TACE
總計	21	385	83	83	450
97年stage1	3	60	14	13	28
97年stage2	0	30	10	8	33
97年stage3	1	31	2	0	49
97年stage4	0	11	0	1	25
98年stage1	3	73	13	26	36
98年stage2	2	38	11	12	62
98年stage3	0	31	0	3	58
98年stage4	0	4	0	1	19
99年stage1	6	60	21	11	28
99年stage2	4	30	10	6	53
99年stage3	0	8	2	2	47
99年stage4	2	9	0	0	12

100年肝細胞癌治療分析
(BCLC於98年開始分析)

	根除性				支持性
	liver transplan	OP	RFA	PEI	TACE
總計	28	105	34	18	146
100年stage1	6	62	27	11	43
100年stage2	12	23	7	4	32
100年stage3	8	14	0	3	61
100年stage4	0	5	0	0	10
不詳	2	1	0	0	0
BCLC A	1	5	6	1	8
BCLC A0	1	11	5	2	4
BCLC A1	1	23	4	6	9
BCLC A2	0	3	7	1	7
BCLC A3	1	1	2	1	8
BCLC A4	1	9	9	2	6
BCLC B	6	25	0	4	46
BCLC C	3	4	0	1	42
BCLC D	8	0	0	0	7
不詳	6	24	1	0	9

LIVER TRANSPLANTATION
MANAGEMENT OF HRS
-EASL CLINICAL PRACTICE GUIDELINES

Liver transplantation is the best treatment for both type 1 and type 2 HRS. HRS should be treated before liver transplantation, since this may improve post-liver transplant outcome (Level A1).

Patients with HRS who respond to vasopressor therapy should be treated by liver transplantation alone. Patients with HRS who do not respond to vasopressor therapy, and who require renal support should generally be treated by liver transplantation alone, since the majority will achieve a recovery of renal function post-liver transplantation. There is a subgroup of patients who require prolonged renal support (>12 weeks), and it is this group that should be considered for combined liver and kidney transplantation (Level B2).

Journal of Hepatology 2010 ; 53 : 397-417.

Thanks for your attention