Better prediction using the modified RECIST compared with the RECIST1.1 with HCC treated with TARE

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Background/Aims: The RECIST 1.1 have been commonly used for response evaluation of solid tumours including HCC. Because of the importance of the remaining 'viable cancer tissue' of HCC, mRECIST have been adopted for HCC response evaluation. However, few studies have investigated which response evaluation method is better for predicting the treatment outcomes of HCC. Thus, we compared the performance of the RECIST 1.1 and mRECIST for predicting survival rates in patients with HCC treated with TARE using Yttrium-90. **Methods:** Between 2012 and 2017, 102 patients with unresectable intrahepatic HCC treated with TARE were reviewed retrospectively. The RECIST 1.1 and mRECIST were used to evaluate the treatment responsiveness of HCC to TARE at 1, 3 and 6 months after TARE. A responder was defined as the sum of either a complete or partial response by each method. **Results:** The median age of the study patients (92 males and 19 females) was 64.1 years. The median AFP and PIVKA-II levels were 42.0 ng/mL and 1693.5 mAU/mL, respectively. The median maximal tumor size was 8.3 cm, and multiple tumours were observed in 41 (36.3%) patients. During the follow-up period (median 20.7 months), 21 patients (20.6%) died, with a median survival of 32.5 months. Using the mRECIST, the treatment responders at 1, 3 and 6 months following TARE showed significantly better survival rates compared with the non-responders (hazard ratio [HR]=5.736, log-rank p=0.008 at 1 month; HR=3.145, p=0.022 at 3 months, and HR=2.887, p=0.061 at 6 months). In contrast, using the RECIST 1.1, the treatment responders at 1, 3 and 6 months In contrast, using the RECIST, the treatment responders at 1, 3, and 6 months showed no statistical difference in survival from the non-responders (all p>0.05, log-rank test). According to multivariate analysis, non-responsiveness using mRECIST (HR=0.217, p=0.043 at 1 month; HR=2.874, p=0.05 at 3 months), as well as the serum albumin level, main portal vein thrombosis, and hepatic vein invasion, were independent predictors of mortality (all p<0.05). **Conclusions:** Risk stratification should be assessed by response evaluation using mRECIST in patients with HCC treated with TARE. Further validation in a large cohort is required.

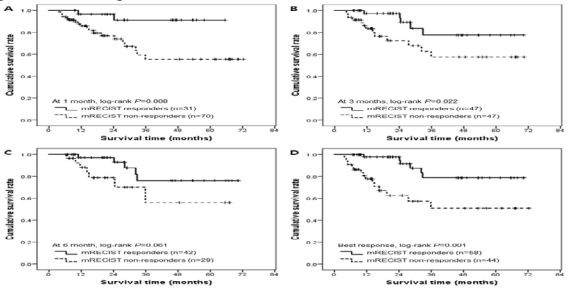

Table 1. Baseline characteristics (n=102)

Variables	Values
Demographic variables	
Age, years	64.3 (53.6-72.8)
Male gender	83 (81.4)
Body mass index, kg/m²	23.9 (21.3-25.8)
Diabetes mellitus	44 (43.1)
Hypertension	59 (57.8)
Viral etiology	71 (69.6)
Liver cirrhosis	28 (27.5)
Laboratory variables	
Platelet count, x10⁹/L	198.5 (121.3-261.5)
Total bilirubin, mg/dL	0.6 (0.5-0.9)
Serum albumin, g/dL	3.9 (3.5-4.1)
Aspartate aminotransferase, IU/L	40.0 (28.8-64.3)
Alanine aminotransferase, IU/L	28.5 (18.0-45.0)
Alkaline phosphatase, IU/L	95.5 (76.0-138.5)
Prothrombin time, INR	1.02 (0.96-1.10)
AFP, ng/mL	37.11 (5.0-2590.1)
DCP, mAU/mL	1780.0 (135.5-8119.5)
Tumor characteristics	
Nodular/ infiltrative	82 (80.4)/ 20 (19.6)
Maximum tumor size, cm	8.3 (6.0-10.5)
Multiple tumors	36 (35.3)
Above three tumors	26 (25.5)
Tumor burden > 50%	10 (9.8)
Bilobar distribution	28 (27.5)
Portal vein thrombosis	19 (18.6)
Hepatic vein invasion	5 (4.9)

Variables are expressed as median (interquartile range) or n (%).
INR, international normalized ratio; AFP, alpha-fetoprotein; DCP, des-gamma-carboxyprothrombin.