Can we apply Baveno-VII criteria in our real-world practice?

AUTHORS:

WONG Yu Jun¹ ², MD; ORCID: 0000-0002-0727-1183

Sanchit SHARMA³, MD; ORCID: 0000-0002-3204-3721

Giulia TOSETTI⁴, MD; ORCID: 0000-0001-5376-9575

#QI Xiaolong⁵, PhD; ORCID: 0000-0002-3559-5855

#Massimo PRIMIGNANI⁴, MRCP; ORCID: 0000-0003-1588-2643

#Co-last authors

AFFILIATIONS:

1. Department of Gastroenterology & Hepatology, Changi General Hospital, Singapore
2. Duke-NUS Academic Clinical Program, SingHealth, Singapore
3. Department of Gastroenterology and Human Nutrition Unit, All India Institute of Medical Science, India
4. Division of Gastroenterology and Hepatology, Foundation IRCCS Ca' Granda Ospedale Maggiore Policlinico, Milan, Italy
5. CHESS Center, Institute of Portal Hypertension, the First Hospital of Lanzhou University, Lanzhou, China

WORD COUNTS: 403

REFERENCES: 6
CORRESPONDING AUTHOR:
Dr Wong Yu Jun, MD, MMed, MCI, FRCP, FAMS
Department of Gastroenterology & Hepatology
Changi General Hospital, Singapore
Email: eugene.wong.y.j@singhealth.com.sg
ORCID: 0000-0002-0727-1183

FUNDING SUPPORT:
W.Y.J was supported by research grant from Medicine Academic Medical Center, Duke-NUS Medical School, Singapore

CONFLICT OF INTEREST:
W.Y.J is an invited speaker for Gilead Science and AbbVie.

AUTHOR CONTRIBUTIONS:
Drafting of manuscript: WYJ, Critical review of manuscript: All authors
CORRESPONDENCE

We thank Dr Semmler and colleague for the interest in our study [1]. In this study, we sought to demonstrate the applicability of NIT-based criteria in risk-stratifying compensated cirrhosis patients in the real-world clinical practice.

We included NASH cirrhosis patients in whom etiological cure is not currently available – these patients represent cACLD without removal of primary etiology in our study. We also included viral-related cirrhosis with adequate virological suppression, which is the current standard of care [2, 3]. The inclusion of treated viral-related cirrhosis should not invalidate our conclusion because even after virological suppression, cirrhosis patients with CSPH may remained to have CSPH, hence these patients remained at risk of future decompensation and hepatocellular carcinoma [1, 4].

While the number-needed to treat is probably higher by including patients with un-treated viral-related cirrhosis, there should be little argument not to treat these patients, given the robust scientific evidences of removing primary etiology to improve outcomes in viral-related cirrhosis patients, as shown in our earlier study [5]. What remains uncertain is whether NSBB are needed to prevent decompensation in all cACLD patients with virological suppression and persistence CSPH - when assessed using NIT-based criteria? [6] Indeed, the risk of first hepatic decompensation was almost half in our patients (predominantly cured HCV infection and CSPH-ruled in by NIT) vs the placebo group of the PREDESCI trial, which included mostly untreated HCV patients with CSPH (13.3% vs 24.0%). This difference likely reflects the impact of virological suppression in HCV patients in our cohort. In a way, it is reassuring to see that NIT-based assessment of CSPH remained predictive of liver decompensation in viral-related cirrhosis patients achieving viral suppression. Because cirrhosis patients may remained to have CSPH (thus the risk of liver decompensation), NSBB should be considered to prevent decompensation in these patients.
As described in our manuscript, patients with NSBB at baseline (presumably higher baseline risk of CSPH, high-risk varices and liver decompensation) were excluded because NSBB may reduce the risk of liver decompensation [7], as shown in PREDESCI trial [8]. Nevertheless, this subgroup is small, and subgroup analysis showed that CSPH (LSM≥25kPa) remained predictive of decompensation after excluding patients with high-risk varices.

There were a significant proportion of patients falling within the grey zone, which was also demonstrated in a recent study by the author [9]. One should not be surprise because this is consistent with the performance of transient elastography to exclude or include patients with advanced fibrosis. While interesting, unfortunately we do not have data on spleen stiffness and the ratio of von Willebrand factor and platelet count (VITRO) in the current cohort.

Finally, as stated in our manuscript, we performed competing risk regression by cluster to account for heterogeneity and regional differences across the four cohorts of patients.

In summary, our findings demonstrated that Baveno-VII criteria of CSPH is predictive of liver decompensation and liver-related events in compensated cirrhosis/cACLD patients. We agree that a pragmatic “non-invasive” PREDESCI trial to re-ensure our current clinical practice using contemporary patients, particularly cACLD patients after HCV cure, would be desirable to confirm these findings. Until then, our findings suggest NSBB should be considered in cirrhosis patients with CSPH diagnosed using non-invasive criteria.


