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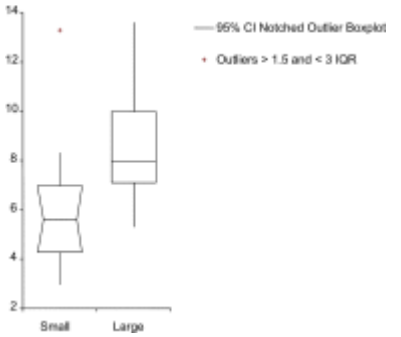
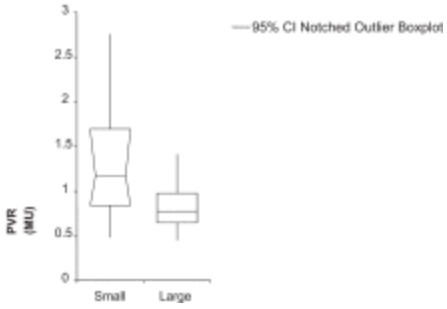
Article outline

Figures and tables

2. 

3. 

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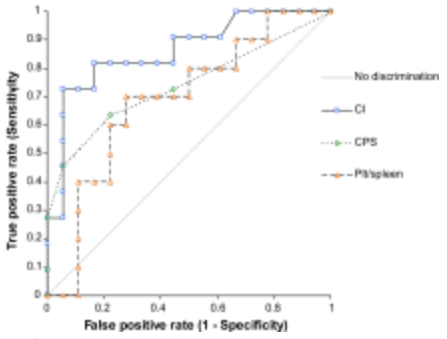
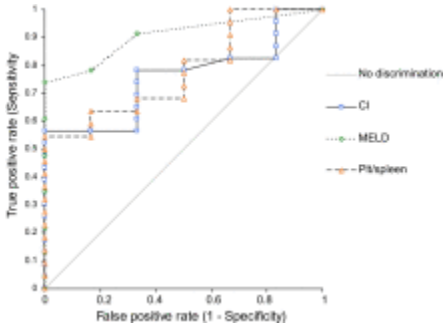
[Table 2]

5. 

6. 

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[Table 3]

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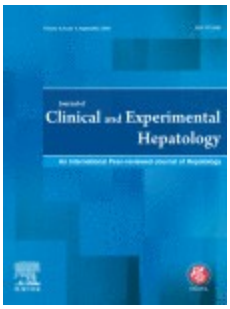

Original Article

Non-invasive Diagnosis of Oesophageal Varices Using Systemic Haemodynamic Measurements by Finometry: Comparison with Other Non-invasive Predictive Scores

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Background/Aims

Cirrhosis and portal hypertension are characterised by a hyperdynamic circulation, which is independently associated with variceal size. Non-invasive techniques for measurement of systemic haemodynamics are now available.

The aim of the study was to prospectively assess the accuracy of systemic haemodynamics measured non-invasively for the detection of oesophageal varices in cirrhotic patients as compared to other currently available non-invasive methods.

Methods

In a study of 29 cirrhotic patients, systemic haemodynamics were studied non-invasively using the Finometer® (mean arterial pressure (MAP), cardiac output (CO)/index, heart rate (HR), peripheral vascular resistance) and portal pressure was assessed by hepatic venous pressure gradient. Sensitivity, specificity, predictive values and area under the receiver operating characteristic (ROC) curves were assessed for predicting presence of varices and large oesophageal varices. Results were compared to child's classification, platelet/spleen ratio and ALT/AST ratios as predictors of the presence of large varices.

Results

Using finometry large oesophageal varices were correctly predicted in 83% of patients compared to other non-invasive techniques (range 66–76%).

Conclusions

Non-invasive assessment of systemic haemodynamics using finometry could aid the identification of patients who do not immediately require variceal surveillance reducing the numbers of endoscopies and ensuring services are provided to those most likely to benefit.

Abbreviations

- MELD, model of end stage liver disease;
- LOV, large oesophageal varices;
- SBP, systolic blood pressure;
- DBP, diastolic blood pressure;
- MAP, mean arterial pressure;
- HR, heart rate;
- SV, stroke volume;
- CO, cardiac output;
- CI, cardiac index;
- PVR, peripheral resistance;
- NIEC, North Italian Endoscopy Club;
- HVPG, hepatic venous pressure gradient;
- PT, prothrombin time;
- AAR, AST/ALT ratio;
- PSDR, platelet count-to spleen diameter ratio;
- IQR, interquartile range;
- ROC, receiver operating characteristic;
- Se, sensitivity;
- Sp, specificity;
- PPV, positive predictive value;
- NPV, negative predictive value;
- LR+, positive likelihood ratio;
- LR−, negative likelihood ratio

Keywords

- oesophageal varices;
- systemic haemodynamics;
- finometry;
- non-invasive predictive scores

Introduction

Although mortality from a variceal bleeding episode has decreased with improved endoscopic and radiological techniques together with new pharmacologic therapies, a 15–20% mortality[1, 2, 3 and 4] means that bleeding from oesophageal varices remains of significant clinical importance. Early diagnosis of varices before the first bleed is essential as studies of primary prophylaxis clearly show that the risk of variceal haemorrhage can be reduced by 50% to about 15% for large oesophageal varices.[5 and 6] Current guidelines therefore recommend that all cirrhotic patients should be screened for varices at diagnosis, with follow-up every 2–3 years for patients without varices (depending upon liver disease severity) and 1–2 yearly for patients with small varices, to assess for enlargement of varices and need for prophylactic treatment.[7] Upper GI endoscopy remains the gold standard for screening, but this test is not without its own limitations. The current guidelines cause a significant burden and cost to endoscopy units, and necessitate patients having repeated unpleasant procedures even when up to 50% may still not have developed oesophageal varices 10 years after the initial diagnosis.[8] If it were possible to predict oesophageal varices by non-invasive means, this restricts testing to the population deemed to be at most risk and reduce the number of endoscopies required. Such a screening test should be simple, quick, reproducible and cost-effective.

Numerous surrogate markers have been evaluated to non-invasively predict the presence of oesophageal varices. These include platelet count (9–12), platelet count/spleen diameter ratio (13–19) and AST/ALT ratio (20). To date, none of these have proved accurate enough to be used routinely and avoid endoscopy.

Portal hypertension is characterised by an increased cardiac output (CO), heart rate (HR) and stroke volume (SV) and reduced peripheral vascular resistance.[21] The Finometer® (Finapres Medical Systems, Amsterdam, The Netherlands) is a non-invasive device that allows continuous beat-to-beat blood pressure and haemodynamic monitoring over a number of hours. Utilising a volume-clamp method to provide a continuous measure of finger pressure with subsequent reconstruction of brachial pressure, it allows the computation of an aortic flow wave form and impedance from which HR, SV, peripheral vascular resistance and cardiac output can be derived. The Finometer therefore provides a non-invasive method of continuous beat-to-beat measurement of systemic haemodynamic variables with good positive correlation to portal pressure.[22]

The aim of this prospective study was to assess the predictive value of systemic haemodynamics assessed non-invasively using finometry for the diagnosis of oesophageal varices in cirrhotic patients, and to compare it with other currently available non-invasive predictors.

Methodology

29 patients with proven cirrhosis (irrespective of aetiology) were studied. All patients were known to have endoscopically proven oesophageal varices or to require an endoscopy for suspected oesophageal varices. Exclusions included a prior history of variceal bleeding requiring therapeutic variceal intervention or known portal vein thrombosis or a history of cardiac complications due to cirrhosis. Non-portal hypertension exclusions included a documented history of cardiac disease or hypertension. Patients with known oesophageal varices taking β-blockers had their drugs discontinued 14 days prior to any haemodynamic measurements. Written consent was obtained and our local research ethics committee approved the study.

The study protocol entailed two separate visits. At the initial visit, endoscopic and non-invasive haemodynamic (fasting) assessments were performed following a detailed clinical and alcohol assessments. Laboratory assessment including bilirubin, albumin, platelet count, prothrombin time (PT), AST, ALT and sodium were undertaken to allow the calculation of the Child–Pugh and MELD scores. At the second visit, a non-fasting non-invasive haemodynamic study was repeated to verify the initial results together with an ultrasound to assess bipolar spleen size.

Evaluation of beat-to-beat blood pressure using the Finometer® was carried out as previously described.[22] Readings were taken on different days and at different times by a single operator trained in the technique. The results of the recordings were not known prior to endoscopy. The following haemodynamic variables were calculated: systolic and diastolic blood pressure (SBP, DBP), mean arterial pressure (MAP), HR, SV, CO, cardiac index (CI), and peripheral resistance (PVR).

An experienced endoscopist performed gastroscopy with oesophageal varices being classified according to the Japanese Research Society for Portal Hypertension and the Japanese score, NIEC index and 1 year probability of bleeding calculated.[23], [24] and [25] Patients were separated into two groups, Group 1 – absent or small varices; Group 2 – medium or large varices, or gastric varices (LOV). The findings were agreed between the endoscopist and a second clinician observing the procedure.

Portal pressure was assessed by measurement of the hepatic venous pressure gradient (HVPG) as described by Groszmann and Wongcharatrawee.[26] HVPG was calculated as the difference between the occluded and the free hepatic venous pressure (mmHg). Three consecutive measurements were taken and the results averaged.

To facilitate the calculation of non-invasive predictive scores, the following laboratory parameters were recorded: serum bilirubin, albumin, PT, platelet count, aspartate transaminase (AST), alanine transaminase (ALT) and creatinine, together with the ultrasound derived bipolar spleen diameter, grade of ascites and encephalopathy. This facilitated calculation of Child–Pugh

score and class, MELD, AST/ALT ratio (AAR) and the platelet count-to spleen diameter ratio (PSDR).

Statistical analysis

Results are expressed as median (interquartile range, IQR). Differences between groups were compared using the Mann–Whitney test and the association between two variables was assessed by the Spearman correlation co-efficient. Analyse-It for Microsoft Excel (Version 2.21) was used for statistical analysis. *P* values <0.05 were considered statistically significant.

Receiver operating characteristic (ROC) curves were constructed and sensitivity (Se), specificity (Sp), positive and negative predictive values (PPV and NPV), positive likelihood ratio (LR+) and negative likelihood ratio (LR−) were calculated. ROC curves were used to establish the optimal cut-offs for each predictor, as they were not specifically designed to detect oesophageal varices.

Results

The characteristics of the 29 patients (20 men and 9 women) are shown in Table 1. The median age was 47 years (42–55 years). Aetiology of the cirrhotic cohort included alcohol abuse (*n* = 18; [62%]), alcohol abuse and hepatitis C (*n* = 5; [17%]), alcohol abuse and autoimmune hepatitis (*n* = 3; [10%]), autoimmune hepatitis (*n* = 2; [7%]) and hepatitis C (*n* = 1; [4%]). 19 patients (66%) were abstinent from alcohol. Child–Pugh stratification was class A (62%), Class B (34%) and Class C (4%). Oesophageal varices were present in 23 patients (79%), classified as small in 12 patients, medium in 8 patients and large in 3 patients. None of the included patients had evidence of gastric varices at endoscopy. As a whole, 18 patients belonged to Group 1 (absent/small varices) and 11 patients to Group 2 (medium/large varices). Ascites was present in 17% of patients (*n* = 5).

Table 1.

Baseline Characteristics of the 29 Cirrhotic Patients, and Univariate Predictors of Large Oesophageal Varices.

	Total *n* = 29	**Group 1 (no OV/small OV) *n* = 18**	**Group 2 (large OV) *n* = 11**	***P* value**
Age (years)	47 (42–55)			
CP score	6 (5–7)	**5 (5–6)**	**7 (5–9)**	**0.023**
MELD	10 (8–13)	10 (7–12)	11 (8–14)	0.17
BMI (kg/m^2)	25.4 (22.5–29.0)			
SBP (mmHg)	147 (131–163)	145 (129–161)	150 (138–163)	0.47
DBP (mmHg)	83 (75–91)	83 (76–91)	79 (73–89)	0.21
MAP (mmHg)	108 (96–	106 (96–116)	112 (95–116)	0.79

	Total $n = 29$	Group 1 (no OV/small OV) $n = 18$	Group 2 (large OV) $n = 11$	*P* value
	115)			
HR (bpm)	80 (71–91)	75 (67–90)	85 (75–93)	0.12
SV (ml)	89 (58–104)	85 (52–101)	91 (85–116)	0.15
CO (lpm)	6.9 (4.8–8.3)	**5.59 (4.29–6.98)**	**7.95 (7.12–10.0)**	**0.003**
CI (l/min/m^2)	3.5 (2.9–4.2)	**3.0 (2.6–3.6)**	**4.5 (3.7–5.5)**	**0.001**
PVR (MU)	0.96 (0.72–1.34)	**1.2 (0.8–1.7)**	**0.8 (0.6–1.0)**	**0.018**
HVPG (mmHg)	17 (11–19)	**14 (10–17)**	**19 (17–21)**	**0.008**
ALT (U/L)	33 (21–49)	38 (22–55)	30 (16–39)	0.192
AST (U/L)	53 (36–74)	53 (37–80)	49 (30–63)	0.301
Platelets ×10^9/L	115 (75–158)	116 (77–160)	97 (71–154)	0.418
PT (s)	13 (12–13)	12 (12–13)	13 (13–14)	0.060
Albumin (g/L)	35 (32–40)	37 (31–41)	33 (32–37)	0.250
Bilirubin (µmol/L)	25 (18–41)	24 (14–40)	27 (21–49)	0.290
Sodium (mmol/L)	139 (136–141)			
AST/ALT ratio	1.6 (1.1–2.1)	1.35 (1.10–1.72)	2.00 (1.55–2.10)	0.105
Platelet count/spleen diameter ratio	833 (488–1276)	973 (631–1438)	569 (407–1023)	0.103

Bold type indicates significant values.

Table options

Table 1 shows the clinical, biochemical and haemodynamic characteristics of the patients according to the size of oesophageal varices. Significant positive correlations were seen between CI, HR and HVPG and disease severity as assessed by Child–Pugh score ($r = 0.36$, $r = 0.37$ and $r = 0.58$ respectively). As there was only a single Child–Pugh class C patient recruited, this was not felt to be representative of the group and was therefore excluded from this part of the analysis. Significant differences in CI (3.3 vs 4.7 l/min/m^2, $P = 0.03$) and HVPG (15 vs 19 mmHg, $P = 0.02$) were seen between Child–Pugh class A and B cirrhotics, together with a significantly prolonged PT (12 vs 13 s, P −0.0007), higher bilirubin (20 vs 42, $P < 0.0001$) and lower albumin (38 vs 32, $P = 0.005$) with worsening liver disease severity.

Using univariate analysis of the variables listed in Table 1 revealed Child–Pugh score, CO, CI, PVR and HVPG showed significant associations with oesophageal variceal size. The median CO was 7.95 lpm in patients with LOV and 5.59 lpm in patients with absent or small oesophageal varices ($P = 0.003$) and the median PVR in patients with LOV was 1.2 MU compared to 0.8 MU

in patients with absent/small oesophageal varices ($P = 0.018$) (Figure 1 and Figure 2). No significant differences were seen in laboratory parameters or predictive scores and variceal size. The ROC curve values for haemodynamic values, laboratory parameters and predictive scores in predicting the presence of oesophageal varices or LOV are shown in Table 2.

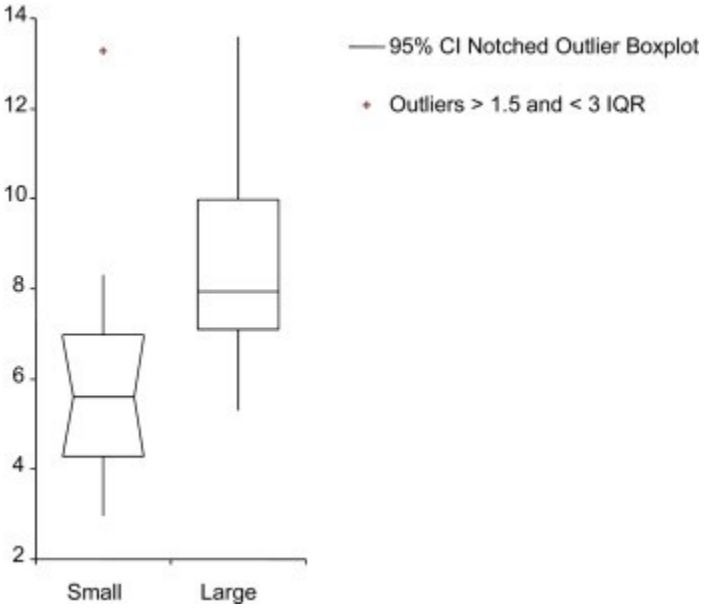

Figure 1.

Boxplot illustrating the relationship between cardiac output and size of oesophageal varices.

Figure options

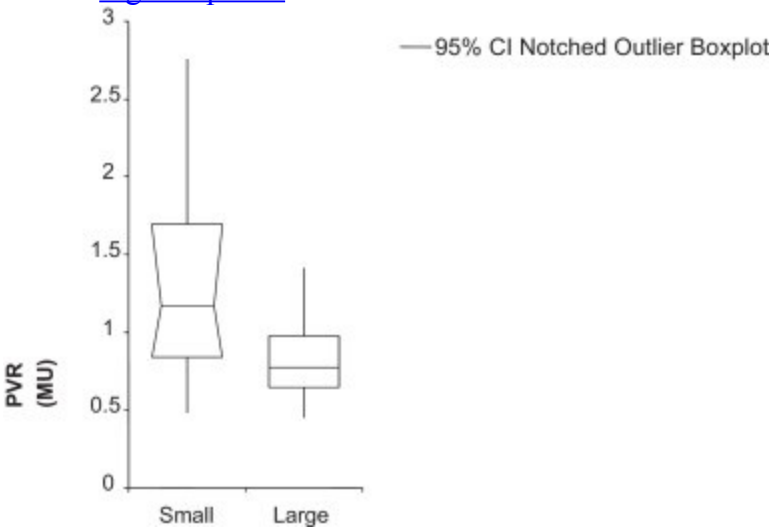

Figure 2.

Boxplot illustrating the relationship between peripheral vascular resistance and size of oesophageal varices.

Figure options

Table 2.

Comparison of AUROC (95% CI) of Different Non-invasive Methods for Discriminating Presence of OV (None vs Any Grade OV) and Presence of Large OV (None/small vs Medium/large OV) in 29 Cirrhotic Patients.

	Presence of OV (*r*)	*P* value	Presence of large OV (*r*)	*P* value
HR	0.78 (0.49–1.00)	0.031	0.68 (0.48–0.87)	0.040
CO	0.71 (0.49–0.92)	0.029	0.84 (0.69–0.99)	<0.001
CI	0.76 (0.51–0.95)	0.004	0.86 (0.71–1.00)	<0.001
PVR	0.63 (0.40–0.86)	ns	0.77 (0.59–0.94)	0.002
BRS	0.81 (0.53–0.96)	0.014	0.81 (0.64–0.98)	ns
HVPG	0.91 (0.74–1.00)	<0.001	0.81 (0.64–0.98)	<0.001
CPS	0.85 (0.75–0.95)	<0.001	0.74 (0.54–0.94)	0.009
MELD	0.91 (0.80–1.00)	<0.001	0.65 (0.45–0.86)	ns
Platelet	0.75 (0.53–0.96)	0.011	0.59 (0.36–0.82)	ns
Albumin	0.32 (0.02–0.61)	ns	0.63 (0.42–0.84)	ns
PT	0.89 (0.79–0.99)	0.015	0.70 (0.52–0.88)	0.015
Bilirubin	0.89 (0.77–1.00)	<0.001	0.62 (0.41–0.83)	ns
AST/ALT ratio	0.73 (0.55–0.91)	0.006	0.68 (0.46–0.91)	ns
Platelet count/spleen diameter ratio	0.78 (0.59–0.97)	0.002	0.69 (0.48–0.90)	0.040

Table options

The ROC curve for CI, Child–Pugh score and PSDR for the prediction of large oesophageal varices are shown in Figure 3. The non-invasive parameters CI and CO yielded the highest AUROC curves (0.86, 0.84). Child–Pugh score, PT and PSDR were the only other non-invasive predictors to have significant AUROC curves. For the presence of large OV, the AUROC curve of CI differed significantly from the AUROC curves for platelet count ($P = 0.036$) and serum albumin ($P = 0.05$). The AUROC curve of PVR for the presence of large OV did not differ from the other parameters. As shown in Figure 4 the AUROC curve for CI, MELD and PSDR for the prediction of the presence of oesophageal varices. MELD, HVPG, PT, bilirubin and Child–Pugh score yielded the highest AUROC curves (0.91, 0.91, 0.89, 0.89, 0.85 respectively). Haemodynamic parameters yielded lower AUROC curves but no significant differences were found between the AUROC curves for CI and any other parameter.

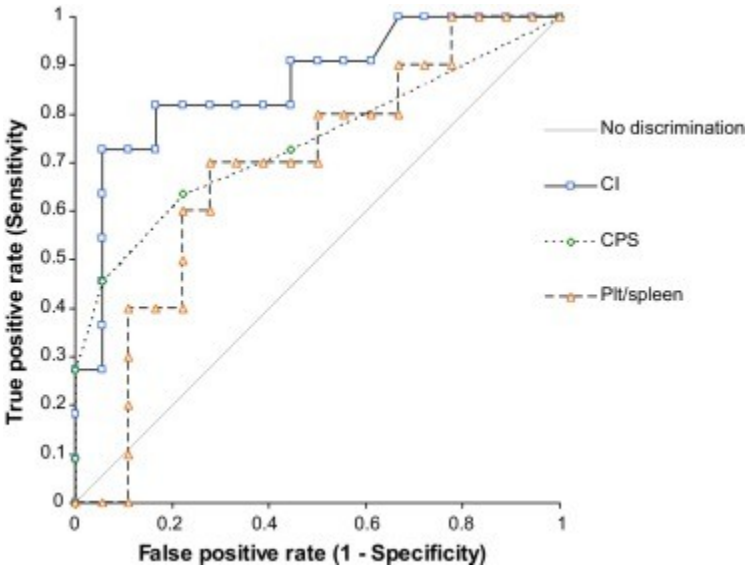

Figure 3.

AUROC curve showing the prediction of large oesophageal varices with cardiac index, Child–Pugh score and platelet count spleen diameter ratio.

Figure options

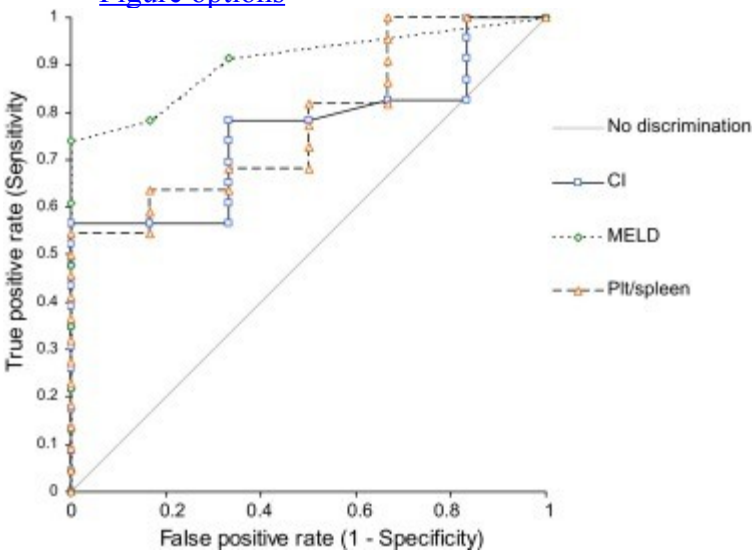

Figure 4.

AUROC curve showing the prediction of presence of oesophageal varices with cardiac index, MELD and platelet count spleen diameter ratio.

Figure options

Diagnostic performances for the presence of LOV are given in Table 3. At a cut-off of 7.06 lpm, CO predicted the presence of LOV with 91% Se and 78% Sp; at a cut-off of 3.66, CI predicted the presence of LOV with 82% Se and 83% Sp; at a cut-off of 0.96 MU, PVR predicted LOV

with 73% Se and 61% Sp. Overall, the percentage of correctly classified patients was as follows: CO (7.06 lpm) 83%, CI (3.66) 83%, AAR 76%, Child–Pugh score 72%, platelet count 69%, PSDR 69%, PT 66%, PVR 66%.

Table 3.

Comparative Performance of Non-invasive Tests for the Diagnosis of Large Oesophageal Varices.

Non-invasive test	Cut-off	Se (%)	Sp (%)	PPV	NPV	LR+	LR−	Patients with large OV *n* = 11	Patients without large OV *n* = 18	Correctly classified
CO	≥5.31	100	44	52	100	1.8	0	11	10	66%
	<5.31							0	8	
	≥7.06									
	<7.06	91	78	71	93	4.09	0.12			86%
								1	14	
	≥7.15									
	<7.15	73	78	67	82	3.27	0.35	8	4	76%
								3	14	
	≥9.15									
	<9.15	46	94	83	74	8.18	0.58	5	1	76%
								6	17	
CI	≥2.85	100	33	48	100	1.5	0	11	12	59%
	<2.85							0	6	
	≥3.12	91	56	56	91	2.05	0.16	10	8	69%
	<3.12							1	10	
	≥3.66	82	83	75	88	4.91	0.22	9	3	83%
	<3.66							2	15	
	≥3.94	73	94	89	85	13.09	0.29	8	1	86%
	<3.94							3	17	
SVR	≤0.78	64	83	70	79	3.52	0.44	7	3	76%
	>0.78							4	15	
	≤0.96	73	61	53	79	1.87	0.45	8	7	66%
	>0.96							3	11	
	≤0.99	91	56	56	91	2.05	0.16	10	8	69%
	>0.99							1	10	
	≤1.41	100	33	48	100	1.50	0	11	12	59%
	>1.41							0	6	

Non-invasive test	Cut-off	Se (%)	Sp (%)	PPV	NPV	LR+	LR−	Patients with large OV *n* = 11	Patients without large OV *n* = 18	Correctly classified
CPS	≥7	64	78	64	78	2.86	0.47	7	4	72%
	<7							4	14	
Platelet count	≤98	64	72	58	77	2.29	0.50	7	5	69%
	>98							4	13	
Platelet count/spleen diameter ratio	≤674	70	72	58	81	2.52	0.42	7	5	69%
	>674							3	13	
AAR	≥1.8	73	78	67	82	3.27	0.35	8	4	76%
	<1.8							3	14	
Prothrombin time	≥13	82	56	53	83	1.84	0.33	9	8	66%
	<13							2	10	

Table options

The optimal cut-offs for haemodynamic parameters remains to be defined. Based on the ROC curves, we looked at the diagnostic values of different cut-offs for the parameters CI, CO, PVR, PT, platelet count, Child–Pugh score, AAR and PSDR for the prediction of large oesophageal varices.

A CO of 5.31 lpm had a Se of 100% and excluded LOV (NPV 100%) but reduced overall accuracy to 66%. A CO of 7.06 l/min improved overall accuracy to 83% with a NPV of 93%; 1 patient in our group would not have been diagnosed with large oesophageal varices at this cut-off. Considering CI, using a cut-off of 3.12 l/min/m^2, this decreased overall accuracy to 69% but improved Se and NPV to 91%; 1 patient in our group would not have been diagnosed with large oesophageal varices at this cut-off. A cut-off of 2.85 l/min/m^2 had a 100% Se and NPV and excluded LOV but overall accuracy was reduced to 59%. Considering PVR, a cut-off of 0.99 improved Se and NPV to 91% and maintained overall accuracy at 69%; 1 patient in this group with large oesophageal varices would not have been identified.

Discussion

This is the first study using systemic haemodynamics, assessed non-invasively to predict the presence of large oesophageal varices and compare with other commonly used non-invasive parameters. As increasing numbers of patients require endoscopic surveillance for varices, increasing interest is being shown in the non-invasive diagnosis of oesophageal varices, due to the cost and burden this places on endoscopy units and the requirement for repeated surveillance over many years. Multiple studies have been performed assessing parameters relating to liver

function, liver fibrosis, portal hypertension and hypersplenism, and combining modalities to produce predictive scores, but to date none of these have proved accurate enough to replace the current gold standard of gastroscopy.[9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19 and 20]

Portal hypertension is the force that drives the development of oesophageal varices in cirrhotic patients. The pathophysiology of portal hypertension is complex but is initiated by increased intra-hepatic resistance and perpetuated by systemic and splanchnic vasodilatation, angiogenesis and remodelling.[21 and 27] The hyperdynamic circulatory syndrome occurs as a result of these changes, correlates with liver disease severity, portal pressure and oesophageal varices[28, 29, 30 and 31] but traditionally requires invasive measurement. Therefore, no studies have assessed the diagnostic ability of systemic haemodynamics to diagnose oesophageal varices. With the development of devices such as the Finometer®, non-invasive continuous beat-to-beat measurement of systemic haemodynamic variables is now possible.[32] We have recently confirmed the ability to detect the hyperdynamic circulation using this technique and shown significant differences in CO according to Child–Pugh class. In addition, we have previously demonstrated that HPVG correlates positively with HR and CI.[22] We have demonstrated significant associations between systemic haemodynamics and HVPG, and 1 year probability of variceal bleeding as assessed by the NIEC index. Non-invasive assessment of systemic haemodynamics in cirrhosis may be useful in predicting HVPG and bleeding risk from oesophageal varices.[33]

As mortality from a first variceal bleed remains high, at 20–30%,[1, 2, 3 and 4] and with the knowledge that primary prophylaxis reduces mortality[5 and 6] in large or high risk oesophageal varices, the importance of detection is clear. The results of this present prospective study indicate that non-invasive assessment of systemic haemodynamics using finometry could be a good predictor of large oesophageal varices in cirrhotic patients. No cut-offs have ever been defined for the diagnosis of large oesophageal varices using systemic haemodynamic parameters but in our study, a cut-off of CO ≥5.31 lpm gave a NPV of 100%. This means that no patients with LOV were missed and that 28% of endoscopies in our study could have been avoided. The diagnostic power of haemodynamics was superior to all other parameters and significantly better than platelet count and serum albumin. A low platelet count is often quoted as being a good predictor of large oesophageal varices but in the studies published to date, there is a wide variation in the cut-off level of platelets used.[9, 10, 11 and 12] Bias is likely to account for much of this variation, with the majority of studies being retrospective in nature, having heterogeneous cohorts of patients, resulting in both selection and spectrum bias. A recent study demonstrated an AUROC curve of 0.62 using a platelet count cut-off of 92,000[12] and our data appear to confirm these results with an AUROC of 0.59.

A study by Giannini et al. combined platelet count with spleen diameter, as assessed by abdominal ultrasound. A cut-off value of 909 was identified, giving a PPV of 96% and NPV of 100%.[13] Subsequently, multiple studies have been performed by other groups with inconsistent results.[14, 15, 16, 17, 18 and 19] Therefore, despite early promise the platelet count/spleen diameter ratio is not a reliable tool to screen for oesophageal varices. Our data would be in agreement with these later studies, with an area under the curve of 0.69 for detection of large oesophageal varices.

Child–Pugh score was the best non-haemodynamic parameter to identify LOV with an area under the curve of 0.74, but using a cut-off of ≥7, this gave a Se of 64% and NPV of 78% and therefore could not be recommended as a non-invasive marker of large oesophageal varices.

The limitations of this study include conditions that would affect systemic haemodynamics, such as the existence of hypertension or prescription of medications that are known to affect systemic haemodynamics, both of which occur frequently in the general population and have the potential to limit the applicability of the test to a wider audience.

Issues have been raised regarding the validity of measurements of absolute arterial blood pressure using the Finometer. A number of studies have shown over- or under-estimation of CO when finometry is compared to the gold standard technique of indicator dilution. However, we feel justified in using finometry as we were not concerned with absolute values per se, but more in assessing the relationships of haemodynamic values to variceal size, relationships that have been demonstrated in other studies using invasive techniques for CO measurement. Model flow is known to be reliable and precise in tracking changes in CO over time in many different patient populations including a small cohort of cirrhotic patients in the above study.[34] and [35] Further studies are needed to track changes in haemodynamics over time and correlate these changes with the change in size of oesophageal varices.

This study is small with only 29 patients, and is comprised of a mixture of aetiologies of liver disease, which may limit the study, but we felt that this represented how the test would be utilised in clinical practice. The group contains very few Child–Pugh C patients, but being primarily composed of Child–Pugh class A and B, it represents the ideal cohort in whom screening and surveillance should be undertaken. However, these results would need to be validated in a larger cohort of patients.

A further limitation is the lack of correlation with transient elastography – a non-invasive technique, which assesses hepatic fibrosis. Numerous studies have been performed assessing its effectiveness to diagnose portal hypertension and oesophageal varices.[36], [37] and [38] Studies comparing transient elastography with finometry are needed.

The advantages of finometry are that it is non-invasive, easily tolerated by patients, reproducible, operator independent and readily performed at the bedside or in the outpatient department. It provides a wealth of information that can be readily downloaded and analysed within a matter of minutes.

In conclusion, the results of this prospective study suggest that in cirrhotic patient's, non-invasive assessment of systemic haemodynamics using finometry may aid identification of patients who do not require variceal surveillance with the result that endoscopic surveillance is provided to those who are most likely to benefit from primary prophylaxis. It also suggests that finometry is of better diagnostic value than other non-invasive markers such as Child–Pugh score or platelet count. Further larger prospective studies are required in order to validate the above findings in all cirrhotics requiring endoscopic surveillance independent of co-morbid disease or prior medication and to track these changes overtime and correlate variceal size.

Conflicts of interest

The authors have none to declare.

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