Correlation between Carotid and Brachial Artery Velocity Time Integral and Their Comparison to Pulse Pressure Variation and Stroke Volume Variation for Assessing Fluid Responsiveness

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ABSTRACT

Background: Fluid boluses are used in hemodynamically unstable patients with presumed hypovolemia, to improve tissue perfusion, in the perioperative period. Now less invasive methods, such as pulse pressure variation (PPV) and stroke volume variation (SVV) are increasingly being used. We investigated correlation between carotid and brachial artery velocity time integral (VTI) and compared both with PPV and SVV. **Methods:** We recruited 27 patients undergoing supra-major abdominal surgeries. When indicated (hypotension or increased lactate), a fluid bolus was given after measuring carotid and brachial artery VTI, PPV, and SVV. The change in SV was noted and patients were categorized as responders if the SV increased by >15%. We performed Bland Altman Agreement and calculated best sensitivity and specificity for the parameters. **Results:** Patients were found to be fluid responders on 29 instances. The correlation between PPV, SVV, carotid and brachial artery VTI was poor and the limits of agreement between them were wide. The Area under Curve (AUC) for PPV was 0.69, for SVV was 0.63, while those of Carotid and Brachial artery VTI (TAP and flow) were (0.53 and 0.54 for carotid) and (0.51 and 0.56 for brachial) respectively.

Conclusion: We found poor agreement and weak correlation between both VTi (TAP and flow) measured at carotid and brachial arteries, suggesting that the readings at brachial vessel cannot be used interchangeably with those at carotid artery. The PPV and SVV were better than these parameters for predicting fluid responsiveness; however, their predictive ability (AUROC), sensitivity and specificity were much lower than previously reported. Further studies in this area are therefore required (CTRI Reg No: CTRI/2017/08/009243).

Keywords: Fluid responders, Fluid responsiveness, Hypoperfusion, Hypovolemia, Pulse pressure variation, Stroke volume variation, Velocity time integral.

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INTRODUCTION

Fluid boluses are used in hemodynamically unstable patients with presumed hypovolemia to improve tissue perfusion. When cardiac index (CI) increases after fluid bolus patients are called responders; this occurs when they are on the steep portion of the Frank Starling curve. However, bedside assessment of fluid responsiveness (FR) remains challenging.¹

Compared to the static measures [pulmonary artery occlusion pressure (PAOP) and central venous pressure (CVP)], dynamic measures such as pulse pressure variation (PPV) and systolic pressure variation (SPV) are better predictors of FR.^{2–5} PPV and SVV measurements need arterial cannulation and these may not predict fluid responsiveness under all conditions.^{1,6} Arterial cannulation itself can cause infections and embolic complications.⁷

Availability of portable ultrasound machines has made noninvasive measurement of hemodynamic parameters, such as aortic blood flow (ABF) or aortic velocity time integral (VTi), possible.⁸ Recently, measurement of carotid and brachial VTi has been shown to be useful for FR prediction.^{9–11} The correlation and degree of agreement between carotid and brachial artery VTi has not been studied before. We therefore evaluated the correlation and agreement between VTI (TAP, time average peak and flow corrected to vessel size) at carotid and brachial arteries ¹Department of Anaesthesiology, Critical Care and Pain, Tata Memorial Hospital, Homi Bhabha National Institute, Mumbai, Maharashtra, India ²Department of Anaesthesia, Critical Care and Pain, Tata Memorial Hospital, Homi Bhabha National Institute, Mumbai, Maharashtra, India ³Department of Critical Care Medicine, Tata Memorial Centre, Homi Bhabha National Institute, Mumbai, Maharashtra, India

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(VTi_TAP_{Carotid}, VTi_TAP_{Brachial}, and VTi_flow_{Brachial} and VTi_ flow_{Carotid}). We also compared the ability of these parameters to PPV and SVV to predict FR in adults.

MATERIALS AND METHODS

We conducted this study after Institutional Ethics Committee (IEC) approval and informed consent. We included adults (>18 years) undergoing elective, supra-major abdominal oncosurgeries under GA. Patients with known peripheral vascular disease, carotid artery disease, history of heart failure, valvular heart disease, and arrhythmias were excluded. Anesthesia technique was standardized. Along with routine monitoring, all patients had their radial artery cannulated, for invasive arterial pressure monitoring. Cardiac output was monitored using FloTrac[™] device with EV1000[™] clinical platform (Edwards Lifesciences, Irvine California, USA). The patient's lungs were ventilated with tidal volume 6 mL/kg predicted body weight, with respiratory rate 12–14 minute, I:E ratio 1:2, and PEEP 5 cm H₂O.

The demographic data, details of surgery, and use of vasopressors (and dose, if used) were recorded. Indications for the fluid challenge were noted. The respiratory (peak and plateau pressures) and hemodynamic parameters (HR, SBP, MAP, CI, SV, PPV, SVV, VTi_TAP_{Carotid}, VTi_TAP_{Brachial}, VTi_flow_{Carotid}, and VTi_flow_{Brachial}) were recorded at baseline, before and after fluid bolus. The tidal volume was transiently increased to 8 mL/kg PBW at the time of observations. Fluid responsiveness was defined as an increase in the SV by 15% in response to fluid administration.

When clinically indicated (hypotension, and/or need for vasopressors, oliguria, lactate levels >2 mmol/L), a fluid bolus of 7 mL/kg Ringers Lactate was given over 30 minutes.

PPV was measured using Philips IntelliVue MP70 monitor. SVV, SV, and CI were recorded using the EV1000[™] clinical platform. A maximum of three sets for all parameters were recorded in every patient, with a gap of at least 2 hours between the two readings.

The Doppler imaging for carotid and brachial artery measurements (VTI TAP i.e., time average peak and flow measured from vessel size by software in machine) were done by a portable ultrasound device, SonoSite Titan HCU (SonoSite; M-TURBO[™], FUJIFILM Sonosite India Pvt. Ltd). A 7–13 MHz broadband linear array transducer was used to obtain carotid and brachial arterial Doppler measurements. The Doppler measurements (VTi_TAP_{Carotid} and VTi_TAP_{Brachial} and VTi_flow_{Carotid} and VTi_flow_{Brachial}) were carried out by a single investigator trained in critical care ultrasound. The brachial artery imaging was performed in the antecubital fossa, on the side without radial artery cannulation. The blood flow velocity was recorded at the midstream of the vessel lumen over 10 seconds with the sample volume adjusted at the center of the artery. The carotid artery velocity waveform was recorded after obtaining a longitudinal view of the common carotid artery, within 2 cm of the bifurcation. The sample volume was positioned at the center of the vessel, with Doppler angulation at approx 20° (not more than 60°). All image angles were corrected up to 15° for the best signal and stored for immediate review following each measurement. An in-built software in the machine computed the VTi_TAP and VTi_flow. The clinician obtaining ultrasound images was blinded to the results of the PPV and SVV, while the clinician recording the PPV and SVV values was blinded to the Doppler results.

Patients were divided into two groups, responders and nonresponders, based on increase in stroke volume (SV) \geq 15%, and we tried to identify the predictors of FR.

Sample Size

This study was meant to be a proof of concept study; we chose a convenience sample size of 50 instances requiring fluid boluses. The fluid boluses were given in 27 patients, when clinically indicated. In nine patients, fluid challenge was required only once, in 13 patients twice, and thrice in five patients.

Statistical Analysis

Data was analyzed using SPSS Statistics version 21 software. Quantitative variables were described using mean \pm SD where data were normally distributed and using median (IQR), where it was skewed. Paired *t* test was used to compare the change in hemodynamic parameters. Student's *t*-test or Mann-Whitney *U* test was used to compare the hemodynamic parameters between responders and nonresponders.

The relation between quantitative variables (VTi_TAP_{Carotid}, VTi_TAP_{Brachial}) was assessed using Pearson and Spearman correlation coefficients according to normality test. Bland-Altman plots were used to describe agreement between VTi_TAP_{Carotid} and VTi_TAP_{Brachial}, and VTi_flow_{Carotid} and VTi_flow_{Brachial}. The receiver operating characteristic (ROC) curves were generated to identify predictors of fluid response and based on highest sensitivity and specificity, cut-offs for PPV, SVV, VTi_TAP_{Carotid} and VTi_TAP_{Brachial}, VTi_flow_{Carotid} and VTi_flow_{Brachial} were determined. All analyses were two-sided, and significance was set at a *p*-value of 0.05.

RESULTS

We included 50 sets of measurements from 27 adults undergoing elective supra-major abdominal surgeries. The mean age of patients was 53 (\pm 20) years and most patients (24) had gastrointestinal malignancies (Table 1). The commonest indications for fluid bolus were hypotension and need for vasopressors (Table 2). Based on the response to fluid bolus, the patients were divided into responders and nonresponders. Table 3 shows the baseline and subsequent (after increasing tidal volume) peak and plateau pressures. Table 4 summarizes the hemodynamic parameters before and after fluid boluses. On 58% occasions, patients were fluid-responsive (Table 4).

We found weak correlation between VTi_TAP_{Carotid} and VTi_TAP_{Brachial} ($r^2 = 0.143$) and VTi_flow_{Carotid} and VTi_flow_{Brachial} ($r^2 = 0.0004$) (Figs 1 and 2). The bias and limits of agreement between the readings taken at carotid and brachial arteries for VTi_TAP and VTi_flow were also wide (Figs 3 and 4). The predictive abilities of PPV and SVV were similar to each other, but low [PPV Receiver Operating Characteristics Curve (AUROC) 0.628 and SVV (AUROC 0.631)]. The AUROC for both VTi_TAP and VTi_flow at carotid and brachial vessels was close to 0.5, suggesting predictive

Table 1: Patients characteristics and	primary diagnosis

	1 , 5
Characteristics	Data (mean <u>+</u> SD)
Age (years)	53 ± 20
Height (cm)	162 <u>+</u> 17
Weight (kg)	59 <u>+</u> 14
BMI	22.9 <u>+</u> 4.8
Females	13
Males	14
Primary diagnosis	No. of patients
Gastrointestinal malignancies	24
Genitourinary malignancies	03



Table 2: Surgical procedures, blood loss, fluid and blood transfusions, and indications for fluid bolus

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Surgical procedures	n
Anterior resection	1
Cytoreductive surgery	2
gynae/genitourinary	
Colorectal cytoreductive surgery with hyperthermic intraperitoneal chemotherapy (CRS + HIPEC)	8
Duodenojejunal flexure resection	1
Colorectal cytoreductive surgery without hyperthermic intraperitoneal chemotherapy (HIPEC)	12
Pylorus preserving pancreatico- duodenectomy (PPPD)	1
Radical cystectomy with ileal Conduit	1
Retroperitoneal lymph node dissection and repair of inferior vena cava (RPLND + IVC repair)	1
Blood loss, fluids and blood and blood	products transfused
Blood loss, mL	2783.33 <u>+</u> 2144.27
Crystalloids, mL	4862.96 <u>+</u> 2944.77
Colloids, mL	1286.11 <u>+</u> 1119.8
PRBCs transfused, no of units (10 patients)	2.18 ± 2.39
FFPs transfused, no of units (8 patients)	2.18 ± 2.39
Indications for fluid bolus [@]	No of patients
Hypotension	10
Lactates >2 mmol/L	04
Need for vasopressors	16
[®] Some patients had more than one indic	ation for fluid bolus

[@]Some patients had more than one indication for fluid bolus

VTi_Flow_{Carotid}

Table 3: Respiratory pressures before and after increasing tidal volume

Peak pressure		Plateau pressure		
Baseline	After increasing	Baseline	After increasing TV	
(6 mL/kg)	TV (8 mL/kg)	(6 mL/kg)	(8 mL/kg)	
19.66 (<u>+</u> 4.15)	19.02 (<u>+</u> 3.71)	15.42 (<u>+</u> 3.77)	15.34 (<u>+</u> 3.42)	

363.47 (147.01)

ability close to a toss of a coin, i.e., very poor (Fig. 5 and Table 5). Highest sensitivity and specificity cut-offs and NPV and PPV for SVV, PPV, VTi_TAP_{Carotid} and VTi_TAP_{brachial}, and VTi_Flow_{carotid} and VTi_Flow_{brachial} were also determined (Table 6).

DISCUSSION

In this prospective observational study we found weak correlation between VTi and VTi_flow (Figs 1 and 2). The bias and LOA between the readings of VTi and VTi_flow at both arteries were also wide (Figs 3 and 4).

We chose to perform this study in patients undergoing elective supra-major oncosurgeries, since during these surgeries, major blood loss and large volume shifts are common. Thus, the patients often become hemodynamically unstable (Table 2). Fast, accurate, and reliable measurement of CO is important for determining FR. Aortic VTi is a well-established method for assessing FR.⁸ However, intraoperative assessment of aortic VTi can be cumbersome in patients undergoing supra-major abdominal surgeries, where the patients are fully draped and the precordial area is difficult to access. It is also difficult to visualize and ensure correct angle of the cardiac ultrasound probe under the drape. Carotid artery Doppler imaging has been shown to be a substitute for the aortic VTI.⁹ However, it also presents difficulty in access, due to drapes and if central venous cannulation has been performed. Carotid and brachial Doppler imaging are easier compared to transthoracic echocardiography, which requires extensive training and is operator-dependent. Monge García et al. showed that measurement of VTi at brachial artery might be a good alternative to aortic VTi.¹⁰ There are no studies evaluating correlation between carotid and brachial artery parameters.

In our study, predictive value of VTi_TAP and VTi_flow at carotid and brachial arteries for FR was poor (Table 7), a finding contrary to previous studies. The AUROC in the study by Monge García et al. was 0.88, and while it was 0.94 in the study by Brenan et al.^{10,11} In both these studies, patients were ventilated with higher tidal volumes: 9 (8–10) mL/kg IBW in the study by Monge Garcia et al., and 9 (\pm 2) mL/kg IBW by Brennan et al.^{10,11}

The low sensitivity and specificity of PPV and SVV to accurately predict fluid responsiveness at the usual cut-offs in our study is not surprising. This may have been due to low tidal volume (TV) ventilation, which is our protocol for all patients undergoing elective

	Nonresponders			Responders		
Variables	Before bolus	After bolus	p value	Before bolus	After bolus	p value
HR (bpm)	79.33 (18.32)	78.24 (16.55)	0.395	90.79 (16.15)	84.55 (16.08)	< 0.001
SBP (mm Hg)	100.59 (19.38)	112.59 (18.91)	<0.001	111.38 (16.21)	121.86 (16.11)	< 0.001
DBP (mm Hg)	60.31 (10.37)	63.55 (10.18)	0.023	65.05 (9.47)	68.24 (8.59)	0.008
MAP (mm Hg)	74.97 (13.72)	81.83 (12.93)	<0.001	82.71 (11.27)	88.38 (10.69)	0.002
Cardiac index	3.39 (0.94)	3.91 (0.86)	<0.001	3.15 (0.72)	3.48 (0.77)	0.002
PPV	14.69 (5.03)	9.86 (4.45)	<0.001	14.1 (5.51)	9.19 (3.28)	< 0.001
SVV	25.91 (12.46)	30.34 (15.98)	0.002	12.57 (3.53)	9 (3.83)	< 0.001
VTi_TAP _{Brachial}	22.59 (9.59)	26.31 (11.38)	<0.001	26.43 (12.90)	30.99 (16.68)	0.027
VTi_Flow _{Brachial}	180.43 (64.72)	220.52 (89.2)	<0.001	204 (75.80)	252.48 (92.30)	< 0.001
VTi_TAP _{Carotid}	17.85 (16.92)	23.74 (19.68)	0.275	14.36 (16.39)	22.70 (19.21)	0.712

T.I.I. A.I.

444.23 (133.44)

HR (bpm), heart rate beats/min; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; SV, stroke volume; PPV, pulse pressure variation; SVV, stroke volume variation; VTI_TAP, velocity time integral averaged peak (carotid or brachial); VTI_Flow, velocity time integral calibrated to vessel diameter (carotid or brachial)

0.002

454.41 (114.09)

0.19

359.37 (126.20)

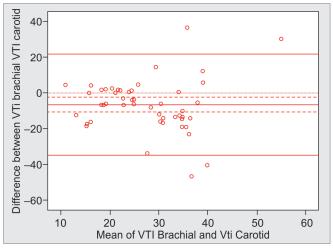


Fig. 1: Correlation between VTI_TAP at carotid and brachial arteries

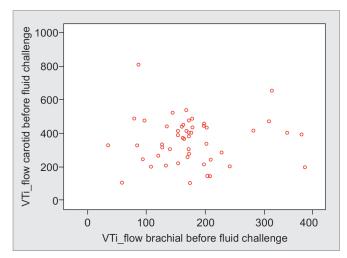
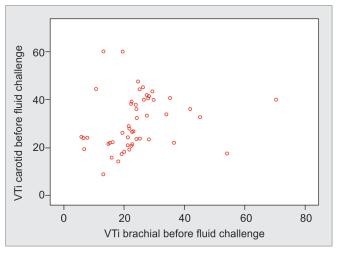
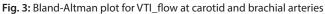


Fig. 2: Correlation between VTI_flow at carotid and brachial arteries





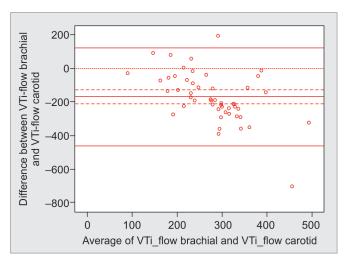


Fig. 4: Bland-Altman plot for VTI_TAP at carotid and brachial arteries

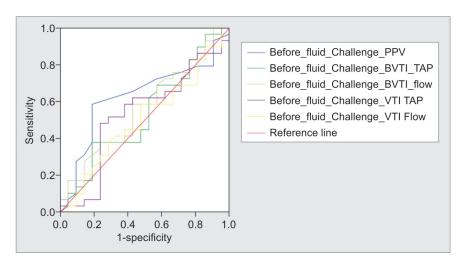


Fig. 5: ROC curves for PPV, SVV, VTI_flow, and VTi_TAP at carotid and brachial vessels



 Table 5: Predictors of patient response to fluid bolus—area under ROC curves

Variable	AUC	95% CI	p value
PPV	0.691	0.468-0.788	0.125
SVV	0.631	0.474-0.787	0.118
VTi_TAP _{Brachial}	0.538	0.373-0.702	0.651
VTi_Flow _{Brachial}	0.567	0.406-0.729	0.420
VTi_TAP _{Carotid}	0.534	0.365-0.702	0.687
VTi_Flow _{Carotid}	0.505	0.341-0.668	0.953

HR (bpm), heart rate beats/minute; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; SVI, stroke volume index; PPV, pulse pressure variation; SVV, stroke volume variation; VTi_TAP_{Brachial}, velocity time integral averaged peak brachial artery; VTi_TAP_{Carotid}, velocity time integral averaged peak carotid artery; VTi_Flow_{Brachial}, velocity time integral calibrated to vessel diameter for brachial artery; VTi_Flow_{Carotid}, velocity time integral calibrated to vessel diameter for carotid artery

Table 6: Predictors of patient response to fluid bolus—area under ROC curves

	Area under		
Variables	ROC curve	95% CI	p value
PPV	0.628	0.468-0.788	0.125
SVV	0.631	0.474-0.787	0.118
VTi_TAP _{Brachial}	0.538	0.373-0.702	0.651
VTi_Flow _{Brachial}	0.567	0.406-0.729	0.420
VTi_TAP _{Carotid}	0.534	0.365-0.702	0.687
VTi_Flow _{Carotid}	0.505	0.341-0.668	0.953

PPV, pulse pressure variation; SVV, stroke volume variation; VTi_TAP_{Brachial}, velocity time integral averaged peak brachial artery; VTi_TAP_{Carotid}, velocity time integral averaged peak carotid artery; VTi_Flow_{Brachial}, velocity time integral calibrated to vessel diameter for brachial artery; VTi_Flow_{Carotid}, velocity time integral calibrated to vessel diameter for carotid artery

Table 7: Sensitivity, specificity, positive, and negative predictive values for PPV, SVV, and VTI_TAP and VTI_flow at carotid and brachial vessels

Cut-off	Sensitivity	Specificity	PPV	NPV
13	75.9%	28.6%	59.5%	46.1%
10	89.7%	19.1%	60.5%	57.1%
10	62.1%	33.3%	56.2%	38.9%
10	70.0%	28.6%	57.1%	40.0%
10	72.4%	28.6%	58.3%	42.9%
10	82.8%	19.0%	58.5%	44.4%
	13 10 10 10 10	13 75.9% 10 89.7% 10 62.1% 10 70.0% 10 72.4%	13 75.9% 28.6% 10 89.7% 19.1% 10 62.1% 33.3% 10 70.0% 28.6% 10 72.4% 28.6%	13 75.9% 28.6% 59.5% 10 89.7% 19.1% 60.5% 10 62.1% 33.3% 56.2% 10 70.0% 28.6% 57.1% 10 72.4% 28.6% 58.3%

supra-major abdominal surgeries. This protocol derives from the current evidence. Futier et al. found that patients ventilated with low TV (6–8 mL/kg PBW) had lower incidence of major pulmonary and extrapulmonary postoperative complications (RR 0.40; 95% Cl 0.24–0.68; p = 0.001) within seven postoperative days, as compared to those ventilated with conventional TV. (10–12 mL/kg PBW).¹² Incidence of ARF requiring NIV or invasive ventilation was lower in low TV group (5.0 vs 17%, RR 0.29; 95% Cl, 0.14–0.61; p = 0.001) and hospital LOS was shorter. There was no difference in mortality. The benefits of intraoperative low TV have been confirmed in three different meta-analyses.^{13–15}

DeBacker et al. demonstrated that PPV performed poorly (51%) in predicting FR at TV of 8 mL/kg IBW as compared with higher volumes (88%).⁶ The performance of PPV was evaluated in a systematic review and meta-analysis of 19 studies (777 patients) ventilated with tidal volumes <8 mL/kg.¹⁶ In 935 fluid challenges, 51.1% patients were found to be fluid responsive. PPV of 10% had the fitted sensitivity of 0.65 (95% CI: 0.57-0.73) to predict FR, and the specificity was 0.79 (95% CI: 0.73–0.84). The AUROC was 0.75. This is similar to our findings for PPV, with sensitivity of 77% and a specificity of 72% but with lower AUROC.¹⁶ The dynamic indices rely on change in intrathoracic pressure. In patients with low lung compliance due to ARDS, the changes in intrathoracic pressure induced by low TV are too small to affect the stroke volume, leading to inability of PPV or SVV to predict fluid responsiveness. We have shown in a previous study that the use of tidal volume challenge (TVC-a transient increase in TV) helps to predict FR.¹⁷ Liu et al. showed that PPV adjusted for change in pleural pressure rather than tidal volume predicted fluid responsiveness better.¹⁸ However, in our study cohort, the patients had normal lungs and there was minimal change in the peak and plateau pressure with increase in TV (Table 3). Using lower cut-offs may offer a solution; however, small errors during measurement might magnify the effects in measurements, affecting the interpretation.⁶

Many studies have tried to investigate if lower cut-offs improve the predictive value of PPV. While studying the effects of tidal volume and adrenergic tone on PPV and aortic VTi, Charron et al. ventilated patients with TV 6–10 mL/kg IBW and varied the TV both ways. The PPV increased with TV both before and after volume expansion. PPV at 10% threshold had sensitivity of 89% and specificity of 83%.¹⁹ Other studies too found improved predictive value of PPV with lower cut-offs (6.5%, 10%).^{20,21} We did not find any improvement in the predictive ability of PPV with the use of lower cut-off in our cohort.

In addition to VTi_TAP, we also studied utility of VTi_flow, to see if the VTi calibrated for vessel diameter makes a difference in predicting FR, which has not been studied before. A small sample size could be a limitation of our study. In our study the fluid boluses were given during acute hypovolemic states and hemodynamic instability. It is possible, therefore, that the fluid bolus given (7 mL/kg) to the patient may have been inadequate to produce the desired response in our patients or they may have responded better to bigger fluid boluses.

CONCLUSION

We found poor agreement and weak correlation between both VTi_TAP and VTi_flow measured at carotid and brachial arteries, suggesting that the readings at brachial vessel cannot be used interchangeably with those at carotid artery. The PPV and SVV were better than these parameters for predicting fluid responsiveness; however, their predictive ability (AUROC), sensitivity, and specificity were much lower than previously reported. Further studies in this area are therefore required.

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184