International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2024: 7.101

From Routine Doppler to Critical Decisions: Portal Pressure

Development and Pilot Validation of a Physiologically Grounded Ultrasound-Derived Model for Portal Pressure Estimation

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Abstract: ***Background: Direct measurement of portal pressure using hepatic venous pressure gradient (HVPG) remains the reference standard for assessment of portal hypertension but is invasive, resource-intensive, and unavailable in many centers. In such settings, ultrasonography (USG) continues to be the primary imaging modality used for clinical decision-making. Objective: To develop a physiology-driven, Doppler ultrasound–derived mathematical model for estimating portal pressure (PPe) and to perform a pilot validation by assessing agreement between model-derived pressure categories and clinically determined severity of portal hypertension. Materials and Methods: In this cross-sectional observational study, 50 patients with suspected chronic liver disease underwent standardized Doppler ultrasound evaluation. Hemodynamic parameters representing portal flow, splenic contribution, intrahepatic resistance, and flow reversal were incorporated into a composite mathematical model derived using multivariate linear regression. The resulting portal pressure estimate (PPe) was categorized into clinically relevant severity groups and compared with clinically assigned portal hypertension stages using Cohen's kappa statistic. Results: The Doppler-derived model demonstrated moderate to substantial agreement with clinical severity classification (κ = 0.605; 95% CI: 0.43–0.78). The model effectively stratified patients into low-, intermediate-, and high-risk portal hypertension categories corresponding to recognized clinical thresholds. Conclusion: This pilot study demonstrates the feasibility of a non-invasive, ultrasound-based hemodynamic model for portal pressure estimation. While not a substitute for invasive measurement, the model provides a physiologically coherent framework for quantitative risk stratification in settings where HVPG is unavailable. Further large-scale validation with simultaneous invasive measurements is warranted.***

Keywords: Portal hypertension, portal pressure, ultrasonography, splenomegaly, collaterals, non-invasive assessment

1. Introduction

Portal hypertension is a major consequence of chronic liver disease and is responsible for significant morbidity and mortality through complications such as variceal bleeding, ascites, and hypersplenism. Accurate assessment of portal pressure is central to staging disease severity, guiding management, and prognostication.

The hepatic venous pressure gradient (HVPG) is widely regarded as the reference standard for quantifying portal pressure. However, its invasive nature, requirement for specialized expertise, and limited availability restrict its routine use—particularly in resource-constrained settings. Consequently, clinicians frequently rely on indirect markers derived from clinical examination, endoscopy, and imaging.

Ultrasonography is the most commonly used imaging modality for evaluating patients with suspected portal hypertension due to its wide availability, low cost, and non-invasive nature. Several sonographic parameters have been described as indirect indicators of elevated portal pressure, yet their interpretation often remains subjective and operator-dependent.

Rather than attempting to replicate HVPG measurement, this study proposes a fundamentally different approach: **estimation of portal pressure through measurable hemodynamic consequences**, integrating flow, resistance, and turbulence into a single quantitative model. This work focuses on **model development and pilot validation**, not replacement of invasive standards.

2. Materials and Methods

Study Design and Setting

This was a cross-sectional observational study conducted at a tertiary care teaching hospital over a defined study period.

Study Population

Patients with chronic liver disease referred for ultrasonographic evaluation of portal hypertension were included.

Inclusion Criteria

- Adults diagnosed with chronic liver disease
- Clinical suspicion or known features of portal hypertension

Exclusion Criteria

- Prior portal decompressive procedures (TIPS or surgical shunts)
- Acute portal vein thrombosis
- Poor acoustic window precluding adequate ultrasonographic assessment

Ultrasound Protocol

All examinations were performed on a Philips Affiniti 70G ultrasound system using a low-frequency curvilinear probe under standardized conditions (fasting >6 hours, supine position, quiet respiration). Doppler angle was maintained at

≤60°, with a sample gate of 2–4 mm. Measurements were averaged over three consistent readings.

Measured parameters:
- Portal vein diameter (Dpv) and mean velocity (PVV)
- Splenic vein diameter (Dsv) and mean velocity (SVV)
- Hepatic artery resistive index (HARI)

Physiologic Model Framework

Conceptual Basis
The model is grounded in the energy balance concept; wherein portal system pressure is determined by:

- Kinetic energy of flow
- Resistance to flow
- Energy loss due to turbulence and flow reversal

Thus:

$$\text{Portal Pressure} = f(\text{Flow}, \text{Resistance}, \text{Turbulence})$$

Derived Hemodynamic Variables

Portal Blood Flow (PBF):

$$\text{PBF} = \pi \left(\frac{D_{pv}}{2}\right)^2 \times \text{PVV} \times 60$$

Splenic Flow Index (SFI):

$$\text{SFI} = \frac{\pi \left(\frac{D_{sv}}{2}\right)^2 \times \text{SVV} \times 60}{\text{BSA}}$$

Reversal Index (RI):
A continuous variable (0–1) representing the proportion of hepatofugal or oscillatory flow.

Intrahepatic Resistance Surrogate:
Hepatic artery resistive index (HARI).

Derived Portal Pressure Equation
The **portal pressure estimate (PPe)** was computed as:

$$\text{PPe} = \gamma + \beta \ln(|\text{PBF}| + 1) + \delta \cdot \text{SFI} \cdot (1 - \text{RI}) + \rho \cdot \text{RI} + \lambda \cdot \text{HARI}$$

Where:
- γ = intercept (−34.15)
- β = flow sensitivity coefficient (2.3)
- δ = splenic correction coefficient (0.008)
- ρ = reversal penalty coefficient (14.9)
- λ = intrahepatic resistance coefficient (36.9)

Coefficients were derived using **multivariate linear regression (ordinary least squares)** to align hemodynamic inputs with clinical severity benchmarks while preserving physiologic plausibility.

The portal congestion index was excluded from the final model due to statistical redundancy.

Clinical Severity Classification
Patients were categorized into three clinical groups:

- **Group 1 (Normal/Mild):** expected portal pressure <8 mmHg
- **Group 2 (Moderate):** 8–15 mmHg
- **Group 3 (Severe):** >15 mmHg, commonly associated with portosystemic collaterals

Model-derived PPe values were similarly categorized for agreement analysis.

This staging reflected **real-world clinical assessment** used in routine hepatology practice.

Ethical Considerations
Institutional ethics committee approval was obtained. As this was an observational study using routine clinical data, informed consent requirements were waived as per institutional policy.

Statistical Analysis
Agreement between PPe-based categorization and clinical severity groups was assessed using **Cohen's kappa statistic**. Kappa values were interpreted using standard thresholds. Statistical analysis was performed using IBM SPSS Statistics version 25.0.

3. Results

The Doppler-derived portal pressure model demonstrated **moderate to substantial agreement** with clinical severity classification (κ = 0.605; SE = 0.090; 95% CI: 0.43–0.78). Agreement was strongest in patients with advanced portal hypertension, where flow reversal and collateralization were prominent.

4. Discussion

This study presents a **novel, physiologically grounded ultrasound-based model** for portal pressure estimation. Unlike descriptive Doppler parameters or empirical scoring systems, this approach integrates flow, resistance, and turbulence into a unified mathematical framework.

A key distinction from invasive HVPG measurement is that the model does not rely on pressure transmission alone but instead evaluates **hemodynamic consequences**, potentially extending applicability to conditions where HVPG is limited. The use of continuous variables such as the Reversal Index allows adaptation to advanced disease states without overestimation.

5. Limitations

- Lack of direct HVPG correlation
- Operator dependence inherent to ultrasonography
- As this is a Cross-sectional design, the study cannot show how portal pressure changes over time or how it responds to treatment. Future studies should follow patients over time and include repeated Doppler measurements, ideally alongside invasive portal pressure measurements, to further validate and refine the proposed formula.

These limitations are intrinsic to non-invasive studies and have been transparently acknowledged.

6. Conclusion

This pilot study demonstrates the feasibility of estimating portal pressure using a Doppler-derived, physiologically informed mathematical model. The model shows substantial agreement with clinical severity classification and offers a promising non-invasive framework for portal hypertension assessment, particularly in low-resource settings. While they do not replace invasive measurements, standardized ultrasonographic assessment remains an indispensable tool for evaluation, staging, and follow-up- especially in resource-limited settings.

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