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# HEMODYNAMIC ALTERATIONS AND BIOCHEMICAL MARKERS OF ENDOGENOUS INTOXICATION IN HEPATIC ECHINOCOCCOSIS: PATHOPHYSIOLOGICAL IMPLICATIONS FOR SURGICAL DECISION-MAKING

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**Abstract**

Hepatic echinococcosis induces progressive and potentially irreversible changes in hepatic parenchymal function and intrahepatic circulation, which vary in severity according to the extent of parasitic involvement and the presence of complications. This thesis examines the relationship between cyst burden, Doppler-assessed portal and hepatic arterial hemodynamics, lipid peroxidation indices, inflammatory biomarkers, and the degree of endogenous intoxication in a cohort of 448 patients with hepatic echinococcosis. Findings demonstrate that cysts exceeding 10 cm in diameter consistently produce measurable disruptions in both portal and arterial flow, and that markers including procalcitonin (PCT), C-reactive protein (CRP), malondialdehyde (MDA), and middle-weight molecules (MWM) correlate strongly with complication severity. These pathophysiological parameters are further advanced as prognostic tools for anticipating organ insufficiency risk and guiding the timing and modality of surgical intervention.

***Keywords:*** *hepatic echinococcosis, portal hemodynamics, Doppler ultrasonography, lipid peroxidation, procalcitonin, C-reactive protein, endogenous intoxication, reactive hepatitis, organ insufficiency*

## 1. Introduction

The pathophysiology of hepatic echinococcosis extends well beyond the mechanical effects of expanding cysts. As parasitic infection progresses, it provokes a cascade of hepatocellular injury, biliary insufficiency, oxidative stress, and systemic inflammatory responses that profoundly alter the patient's clinical trajectory and surgical risk profile. Yet in clinical practice, the pathophysiological dimension of HE has been underweighted relative to morphological and anatomical considerations — with the result that surgical timing and approach selection have often been guided by cyst size and location alone, without systematic assessment of underlying hepatic functional impairment.

This thesis posits that a comprehensive pathophysiological assessment — integrating hepatic hemodynamics, lipid peroxidation indices, acute-phase inflammatory proteins, and endogenous intoxication scores — provides clinically actionable information that should complement imaging-based staging in the preoperative workup of all patients with hepatic echinococcosis. Specifically, it argues that Doppler volumetric flow parameters in the portal venous system constitute reliable predictors of postoperative organ insufficiency, and that the biochemical severity of endogenous intoxication in complicated cases justifies a staged surgical strategy rather than immediate radical intervention.

## 2. Pathophysiological Framework

Liver biopsy specimens obtained intraoperatively and by percutaneous puncture from 58 patients allowed direct assessment of hepatic parenchymal integrity. Diene conjugates (DC) and malondialdehyde (MDA) — surrogate markers of lipid peroxidation — were measured in hepatic tissue and peripheral blood. In patients with bilateral hepatic involvement or complicated echinococcosis, DC values reached 1.78 ± 0.42 and 1.90 ± 0.34 relative units, respectively, against a baseline of 0.9 ± 0.32 in uncomplicated disease. MDA in hepatic tissue averaged 3.2 ± 0.3 μmol/L in bilateral involvement and 3.5 ± 0.5 μmol/L in complicated cases, compared to 2.2 ± 0.2 μmol/L in uncomplicated hepatic echinococcosis. These gradients confirm progressive oxidative hepatocellular damage as parasitic load increases.

Histological examination revealed morphological patterns consistent with reactive hepatitis across the majority of biopsy specimens: lymphocytic infiltration of portal tracts, canalicular and intrahepatic cholestasis in cases complicated by obstructive jaundice, and progressive hepatocyte necrosis in the most severely affected individuals. The de Ritis coefficient — the ratio of aspartate aminotransferase to alanine aminotransferase — confirmed hepatocellular cytolysis in complicated presentations, with statistically significant transaminase elevations correlated to complication type.

## 3. Hemodynamic Findings

Doppler ultrasound examination was performed in all 448 patients. Volumetric hepatic arterial blood flow increased progressively with cyst diameter: 248 mL/min for cysts under 10 cm, 286 mL/min for cysts of 10–15 cm, and 410 mL/min for cysts exceeding 15 cm ($p < 0.05$). These figures likely reflect compensatory arterial hyperperfusion in response to compromised portal inflow — a phenomenon analogous

to the hepatic arterial buffer response described in other space-occupying hepatic lesions.

Intraoperative duplex scanning of the portal vein was performed in 35 cases, comparing hemodynamic parameters between patients with large (> 15 cm) and medium (10–15 cm) cysts. Portal vein diameter, mean velocity, and volumetric flow (1062.3 ± 82.1 vs. 1005.4 ± 76.1 mL/min, respectively, $p < 0.05$) differed significantly between groups, establishing a direct correlation between parasite burden and portal flow impairment. Critically, portal venous flow below 480 mL/min in the early postoperative period was identified as a threshold value predicting heightened risk of organ insufficiency. Values of 700–1120 mL/min and above 1120 mL/min were associated with favorable prognoses.

**4. Endogenous Intoxication and Inflammatory Markers**

The Leukocyte Intoxication Index (LII) averaged 4.4 ± 0.06 units in patients with suppurated hepatic echinococcosis, reflecting significant systemic inflammatory burden. Serum procalcitonin (PCT) reached 3.6 ± 0.02 ng/mL in suppurated cases versus 0.8 ± 0.02 ng/mL in uncomplicated disease, and 5.6 ± 0.04 ng/mL when obstructive jaundice was also present. C-reactive protein (CRP) followed a corresponding pattern: 192.8 ± 15.2 mg/L in suppurated cases versus 6.3 ± 0.8 mg/L in uncomplicated disease, rising to 210.6 ± 9.8 mg/L with concurrent jaundice.

In the most severe presentations (heavy-grade obstructive jaundice), total bilirubin reached 200 µmol/L, MDA rose to 8.16 ± 0.4 µmol/L, and middle-weight molecules (MWM) approached 0.6 units — thresholds indicative of hepatotoxic metabolite accumulation with systemic sequelae. The effective albumin concentration (EAC) was reduced by 21.9% in obstructive jaundice and by 30.4% in suppurated cases relative to uncomplicated disease, signaling impaired hepatic synthetic function. The toxicity index in suppurated cases exceeded that in parasitic jaundice by 18.4% ($p < 0.05$), confirming the heightened danger of septic complications as a specific pathophysiological domain.

**5. Clinical Implications and Conclusion**

The pathophysiological data assembled in this thesis support several clinically significant propositions. First, early surgical intervention — when cysts remain below 5 cm — prevents the progressive hemodynamic, oxidative, and inflammatory deterioration documented in larger or complicated lesions, and minimally invasive methods are particularly well suited to this window of intervention. Second,

preoperative portal Doppler assessment should be incorporated into the standard workup for all patients scheduled for hepatic echinococcosis surgery, since volumetric flow thresholds reliably stratify organ insufficiency risk in the postoperative period. Third, in complicated cases presenting with severe endogenous intoxication — as quantified by PCT, CRP, LII, MDA, and MWM — definitive radical surgery should be deferred until biochemical normalization is achieved through staged minimally invasive septic focus sanitation and supportive hepatoprotective therapy.

In conclusion, pathophysiological evaluation of hepatic hemodynamics and systemic inflammatory burden constitutes an indispensable — though frequently overlooked — dimension of preoperative assessment in hepatic echinococcosis. The integration of Doppler parameters and biochemical intoxication indices into surgical decision algorithms represents a substantive advance toward individualized, evidence-based management of this complex parasitic disease.