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ASSESSMENT OF CENTRAL HEMODYNAMICS IN DIABETES WITH ENDOTOXEMIA

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Abstract
Diabetes mellitus is currently one of the fastest-growing chronic diseases worldwide, continuing to spread at an alarming rate across all regions. According to data from the International Diabetes Federation (IDF), an estimated 537 million people were living with diabetes in 2021, and this number is projected to rise to 783 million by 2045 [1].
The late complications of diabetes, particularly purulent–septic inflammations of the lower limbs — such as diabetic foot syndrome, phlegmon, and gangrene — have a profoundly negative impact on patients' quality of life. These conditions are associated with a markedly increased risk of limb amputation and mortality.

Keywords. Diabetes mellitus; sepsis; endotoxicosis; central hemodynamics; NT-proBNP; lactate; echocardiography; microcirculation; vasoplegia; cardiogenic dysfunction.

Introduction

Annotation

One of the advanced complications of diabetes mellitus is purulent-septic inflammation of the soft tissues of the lower limbs. This condition tends to worsen systemic endotoxicosis and leads to a wide range of hemodynamic disturbances. In this study, we focus on a detailed assessment of these pathological processes through an integrated analysis of central hemodynamic parameters.
From a pathophysiological perspective, this condition is closely linked to endotoxicosis, which contributes to the development of hypovolemic, cardiac,

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vasoplegic, and microcirculatory disturbances. Endotoxic shock and septic cardiomyopathy may lead to multiple organ failure in such patients [2,3]. Hemodynamic disorders in this context are complex and require a thorough, multidimensional assessment because:

- **Hypovolemic dysfunction** is associated with fluid loss resulting from increased vascular permeability in the setting of diabetes [4].
- **Cardiac dysfunction (septic cardiomyopathy)** develops under the influence of endotoxins and inflammatory mediators [5].
- **Vasoplegic dysfunction** occurs due to the excessive synthesis of nitric oxide (NO) [6].
- **Microcirculatory disturbances** impair tissue oxygen delivery and may lead to necrosis [7].

At present, there are no sufficiently reliable or standardized scoring systems for identifying and evaluating hemodynamic disturbances that develop in the context of sepsis and diabetes mellitus. Therefore, a comprehensive and integrative assessment of central hemodynamic parameters represents one of the most relevant scientific approaches for improving diagnosis, treatment, and prognosis of this pathology.

This study is aimed at developing a new diagnostic algorithm through the evaluation of hypovolemic, cardiac, vasoplegic, and microcirculatory disturbances, ultimately contributing to the optimization of clinical decision-making to reduce mortality and disability among patients [8,9]. The present article specifically addresses this issue by identifying central hemodynamic disorders in patients with purulent-septic inflammation on the background of diabetes mellitus and proposing an individualized approach that may serve as the foundation for new scientific and clinical strategies [10,11].

Aim

Therefore, the aim of this study is to develop a comprehensive approach for identifying and evaluating hemodynamic disturbances in patients with purulent-septic inflammation associated with diabetes mellitus, and to design an individualized diagnostic algorithm for their clinical assessment.

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Materials and Methods

The study was conducted in a prospective format and focused on investigating the pathophysiological mechanisms underlying hemodynamic disturbances. The analysis was carried out in a cohort of patients exhibiting endotoxin-induced alterations in central hemodynamics.

Assessed Parameters

- **Hemodynamic indicators:** arterial pressure, cardiac index, total peripheral vascular resistance (TPVR), and central venous pressure (CVP) dynamics.
- **Echocardiographic parameters:** systolic and diastolic ejection fraction, stroke volume, and myocardial contractility.
- **Clinical course dynamics:** stages of hemodynamic disturbances and their progression over time.

Study Participants

- **Total number of patients:** 64
- **Study group:** all 64 patients were included in the analysis, focusing on the evaluation of the pathophysiological mechanisms underlying hemodynamic disturbances.

Inclusion Criteria

Patients were included in the study if they met the following criteria:

- A confirmed diagnosis of sepsis or endotoxemia.
- Presence of at least one of the following indicators:
  - Mean arterial pressure (MAP) < 65 mm Hg.
  - Cardiac index (CI) < 4 L/min.
  - Serum lactate level > 2 mmol/L.
- Clinical signs of hypovolemic, hyperkinetic, or vasoplegic states.
- Echocardiographically verified dynamic alterations in myocardial function.
- Changes in central venous pressure (CVP) and total peripheral vascular resistance (TPVR).
- Age ≥ 18 years, regardless of sex.
- Written informed consent obtained from the patient or their legal representative.

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Exclusion Criteria

Patients were excluded from the study if any of the following conditions were present:

- Severe chronic heart failure, NYHA class III–IV.
- Terminal-stage hepatic or renal failure.
- Decompensated endocrine disorders (e.g., thyrotoxicosis, Addison's disease, etc.).
- Active oncologic or systemic autoimmune diseases.
- Conditions requiring emergency surgery that interfere with hemodynamic assessment.
- Procalcitonin level ≤ 0.5 ng/mL (absence of a clear septic response).
- Severe neuroinfections or primary central nervous system injury.
- Pregnancy or lactation.

Research Methods

- **Hemodynamic monitoring:** performed using both invasive and non-invasive assessment techniques.
- **Laboratory diagnostics:** included the evaluation of inflammatory and endotoxemia markers, along with standard biochemical and hematological tests.
- **Statistical analysis:** data were processed using *SPSS* version 26.0; comparisons were made with *ANOVA* and *t-tests* where appropriate.

Results

During the study, endotoxin-induced hemodynamic disturbances were analyzed in a stage-by-stage manner. The hyperkinetic phase was excluded from the final analysis, as no clinically significant changes were observed in central hemodynamic parameters or biomarkers during this stage. The study therefore focused on the pathophysiological mechanisms and dynamics of hypovolemic, vasogenic, and cardiogenic disturbances.

For mathematical modeling of hemodynamic changes, the mean arterial pressure (MAP) was calculated according to the following formula:

MAP = (SV × TPR) + CVP

In this formula:

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- **MAP (Mean Arterial Pressure)** — the key indicator reflecting the level of tissue perfusion.
- **SV (Stroke Volume)** — a parameter characterizing the heart's pumping function.
- **TPR (Total Peripheral Resistance)** — a determinant of systemic hydrodynamic resistance.
- **CVP (Central Venous Pressure)** — an indicator of venous return and right ventricular preload.

Hemodynamic Disturbances and Key Findings

Throughout the study, major hemodynamic parameters were evaluated across different types of disturbances — hypovolemic, cardiogenic, vasogenic, and microcirculatory. These parameters serve as essential indicators for determining whether systemic circulation is functioning within physiological limits or has shifted toward a pathological state.

Hemodynamic Findings by Disturbance Type

Hypovolemic Disturbances

- **MAP:** 62.1 mm Hg — indicative of hypotension.
- **HR:** 113 bpm — tachycardia.
- **CVP:** 3 mm Hg — reduced venous return.
- **CI:** 3.27 L/min — decreased cardiac output.
- **TPVR:** 1800 dyn·s·cm^{-5} — compensatory vasoconstriction.
- **Lactate:** 3.9 mmol/L — hypoxic metabolic acidosis.
- **NT-proBNP:** 2500 pg/mL.

Parameter	Value	Normal Range	Interpretation
MAP (mm Hg)	62.1	70–100	Moderate hypotension due to reduced circulating blood volume.
HR (beats/min)	113.0	60–90	Compensatory tachycardia aimed at maintaining cardiac output.
CVP (mm Hg)	3.0	5–10	Markedly decreased — indicates venous return and preload deficiency.
CI (L/min)	3.27	4.0–7.0	Slightly reduced — cardiac output decreases with ongoing hypovolemia.
TPVR (dyn·s·cm^{-5})	1800	900–1400	Increased — peripheral vasoconstriction as a compensatory mechanism.
Lactate (mmol/L)	3.9	0.5–2.2	Elevated — early tissue hypoperfusion and oxygen debt.
NT-proBNP (pg/mL)	2500	<125 (elderly) / <400 (young)	Moderately increased — reflects cardiac strain under hypovolemic stress.

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Cardiogenic Disturbances

- **MAP:** 55.0 mm Hg — moderate hypotension.
- **HR:** 110 bpm — reduced cardiac performance.
- **CVP:** 10 mm Hg — elevated due to heart failure.
- **CI:** 1.99 L/min — cardiogenic collapse (observed in fatal cases).
- **TPVR:** 2100 dyn·s·cm^{-5} — peripheral vasoconstriction.
- **Lactate:** 5.2 mmol/L — critical tissue hypoxia (in lethal outcomes).
- **NT-proBNP:** 7500 pg/mL.

MAP (Mean Arterial Pressure)	55.0 mmHg	Moderate hypotension indicating impaired perfusion.
HR (Heart Rate)	110 bpm	Compensatory tachycardia reflecting reduced cardiac output.
CVP (Central Venous Pressure)	10 mmHg	Elevated due to left or right heart failure; venous congestion.
CI (Cardiac Index)	1.99 L/min/m²	Critically low cardiac performance; approaching cardiogenic collapse.
TPVR (Total Peripheral Vascular Resistance)	2100 dyn·s·cm^{-5}	Peripheral vasoconstriction as compensatory response.
Lactate	5.2 mmol/L	Severe tissue hypoxia; marker of inadequate perfusion, associated with lethal outcomes.
NT-proBNP	7500 pg/mL	High level indicating significant myocardial strain and heart failure severity.

Vasogenic Disturbances

- **MAP:** 54.3 mm Hg — pronounced hypotension.
- **HR:** 151.6 bpm — severe tachycardia.
- **CVP:** 8 mm Hg — relatively preserved.
- **CI:** 6.06 L/min — hyperkinetic cardiac state.
- **TPVR:** 600 dyn·s·cm^{-5} — pathological vasodilation.
- **Lactate:** 6.5 mmol/L — metabolic acidosis (in fatal cases).
- **NT-proBNP:** 5600 pg/mL.

Parameter	**Value**	**Comment**
MAP (Mean Arterial Pressure)	54.3 mmHg	Pronounced hypotension reflecting severe systemic vasodilation
HR (Heart Rate)	152 bpm	Marked tachycardia as a compensatory response
CVP (Central Venous Pressure)	8 mmHg	Relatively preserved venous return
CI (Cardiac Index)	6.06 L/min/m²	Hyperkinetic cardiac state
TPVR (Total Peripheral Vascular Resistance)	600 dyn·s·cm^{-5}	Pathological reduction in peripheral resistance
Lactate	6.5 mmol/L	Metabolic acidosis, characteristic of fatal outcomes
NT-proBNP	5600 pg/mL	Elevated, reflecting myocardial strain

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Microcirculatory Type of Hemodynamic Disorder

- **MAP: 58.0 mm Hg** — relative hypotension; systemic pressure near the lower limit of perfusion.
- **HR: 127.9 bpm** — persistent tachycardia reflecting sympathetic overstimulation.
- **CVP: 7 mm Hg** — within near-normal range; venous return preserved but ineffective for tissue perfusion.
- **CI: 4.45 L/min** — within "normal" values, indicating a *pseudo-stable* cardiac output despite tissue hypoxia.
- **TPVR: 700 dyn·s·cm^{-5}** — decreased vascular resistance, consistent with microvascular vasoplegia.
- **Lactate: 7.2 mmol/L** — marked lactic acidosis; indicates severe tissue hypoperfusion and anaerobic metabolism.
- **NT-proBNP: 6800 pg/mL** — elevated myocardial stress marker suggesting ventricular overload and endothelial dysfunction.

Parameter	Value	Comment
MAP (Mean Arterial Pressure)	58.0 mmHg	Relative hypotension; systemic pressure near the lower limit of tissue perfusion
HR (Heart Rate)	127.9 bpm	Persistent tachycardia reflecting sympathetic overstimulation
CVP (Central Venous Pressure)	7 mmHg	Near-normal; venous return preserved but ineffective for tissue perfusion
CI (Cardiac Index)	4.45 L/min/m²	Within nominal range, indicating pseudo-stable cardiac output despite hypoperfusion
TPVR (Total Peripheral Vascular Resistance)	700 dyn·s·cm^{-5}	Reduced vascular resistance consistent with microvascular vasoplegia
Lactate	7.2 mmol/L	Severe lactic acidosis indicating critical tissue hypoperfusion
NT-proBNP	6800 pg/mL	Elevated, reflecting myocardial stress, ventricular overload, and endothelial dysfunction

Comparative Hemodynamic Parameters Across Disturbance Types

Parameter	Hypovolemic	Cardiogenic	Vasogenic	Microcirculatory	Normal Range
MAP (mm Hg)	62.1	55.0	54.3	58.0	70–100 mm Hg
HR (beats/min)	113.0	110.0	151.6	127.9	60–90 beats/min
CVP (mm Hg)	3.0	10.0	8.0	7.0	5–10 mm Hg
CI (L/min)	3.27	1.99	6.06	4.45	4.0–7.0 L/min
TPVR (dyn·s·cm^{-5})	1800.0	2100.0	600.0	700.0	900–1400 dyn·s·cm^{-5}
Lactate (mmol/L)	3.9	5.2	6.5	7.2	0.5–2.2 mmol/L
NT-proBNP (pg/mL)	2500.0	7500.0	5600.0	6800.0	<125 pg/mL (elderly) or <400 pg/mL (younger adults)

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Discussion

The results of the study demonstrated that endotoxin-induced hemodynamic disturbances develop in a distinct sequential pattern. Each stage is characterized by its own specific pathophysiological mechanisms and compensatory responses.

1. Hypovolemic Phase

A marked reduction in stroke volume (SV) and central venous pressure (CVP) was observed during this stage. Total peripheral vascular resistance (TPVR) increased compensatorily, reflecting an attempt to maintain tissue perfusion and stabilize circulation temporarily despite volume loss.

2. Cardiogenic Phase

This stage was characterized by impaired myocardial contractility, leading to a significant decrease in cardiac output (CO). Although peripheral vascular resistance (TPVR) increased as a compensatory response, the overall hemodynamic balance was disrupted due to insufficient cardiac performance.

3. Vasogenic Phase

A progressive decline in total peripheral vascular resistance (TPVR) and mean arterial pressure (MAP) was observed. The loss of vascular tone, primarily induced by inflammatory mediators and excessive nitric oxide production, resulted in pathological vasodilation and redistribution of microcirculatory flow.

4. Microcirculatory Phase

At this stage, capillary perfusion became critically impaired, leading to inadequate oxygen delivery to tissues. Progressive metabolic imbalance and cellular energy deficit developed, contributing to the deterioration of vital physiological processes and eventual multi-organ dysfunction.

Conclusion

1. Echocardiography (ECHO), serum lactate, NT-proBNP, and central venous pressure (CVP) are key diagnostic markers for the comprehensive assessment of central hemodynamic disturbances.
2. Given the multi-stage and dynamic nature of sepsis, evaluation based on a single parameter is insufficient. A combined analysis of central hemodynamic parameters should be considered the optimal diagnostic approach.

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3. Continuous central hemodynamic monitoring is essential for characterizing hypovolemic, cardiogenic, and vasoplegic disturbances; a joint assessment of these parameters helps clarify their underlying pathophysiological mechanisms.
4. The study findings demonstrate that a systematic evaluation of central hemodynamic parameters provides an effective framework for understanding the diverse hemodynamic phenotypes of sepsis.
5. Considering the complex nature of sepsis, an integrated analysis of these parameters contributes to the development of more individualized therapeutic strategies.

These conclusions hold substantial clinical value for improving the evaluation, monitoring, and management of sepsis-related hemodynamic disorders.

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