Letter to Editor

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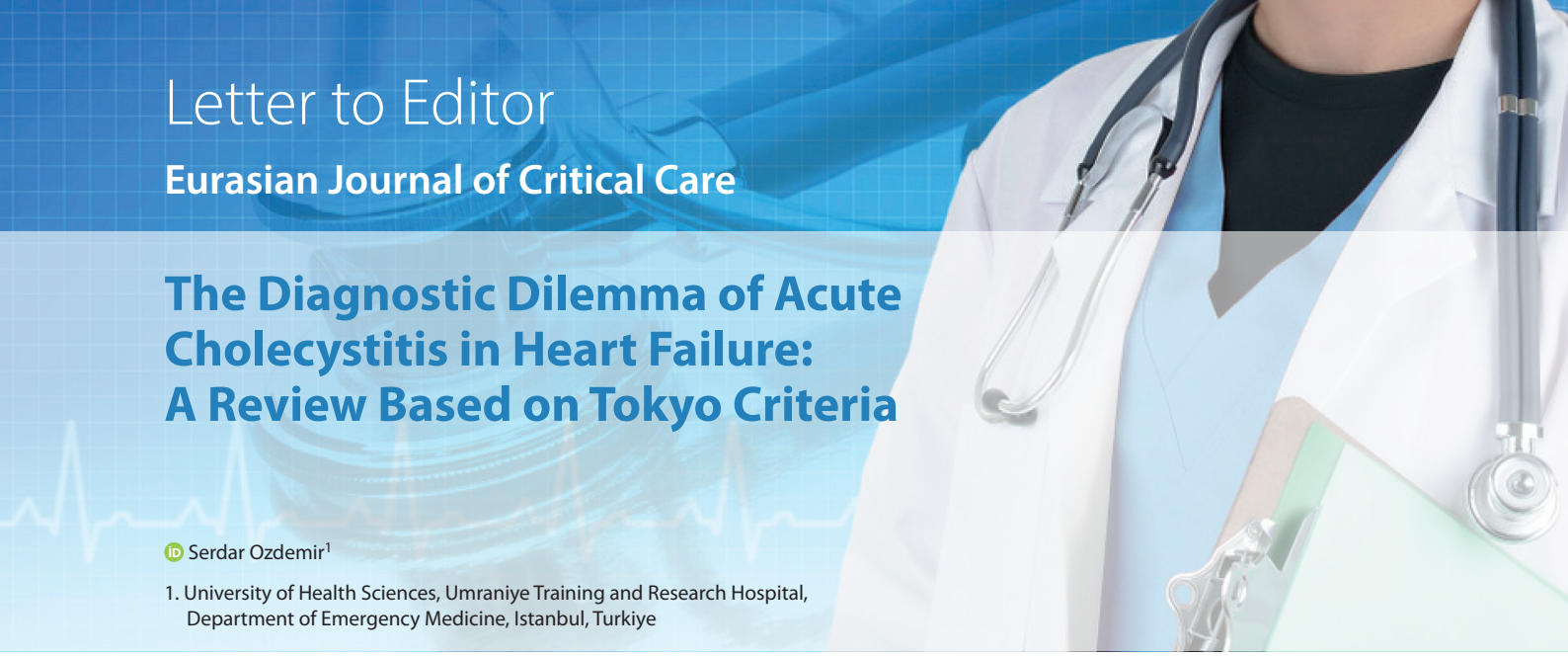

The Diagnostic Dilemma of Acute Cholecystitis in Heart Failure: A Review Based on Tokyo Criteria

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Abstract

Diagnosing acute cholecystitis in patients with heart failure presents a significant clinical challenge due to overlapping symptoms, imaging findings, and inflammatory markers. The Tokyo Guidelines (TG18) provides a structured diagnostic framework based on clinical signs, imaging, and laboratory parameters. However, heart failure—particularly right-sided heart failure—factors such as hepatic congestion, peritoneal stretching, and systemic inflammation can mimic cholecystitis. Gallbladder wall thickening, a key imaging criterion, is often observed in heart failure due to venous congestion rather than true inflammation. Similarly, elevated inflammatory markers like C-reactive protein (CRP) and leukocytosis can result from the systemic inflammatory milieu of heart failure, not infection. This overlap risks both misdiagnosis and inappropriate treatment. This review highlights the diagnostic intersection, emphasizing the importance of a multidisciplinary approach, advanced imaging modalities, and dynamic monitoring. It also advocates for the development of integrated diagnostic tools tailored for this patient population. Accurate differentiation between true acute cholecystitis and heart failure-related changes is essential to avoid unnecessary interventions and optimize patient outcomes.

Keywords: Acute Cholecystitis, Heart Failure, Symptoms

Introduction

Dear Editor,

We appreciate the opportunity to discuss an important clinical conundrum in the diagnosis of acute cholecystitis in patients with heart failure. The Tokyo Guidelines have provided a standardized framework for diagnosing acute cholecystitis, focusing on three main parameters: clinical symptoms, radiologic findings, and inflammatory response.

While these criteria are invaluable in routine cases, their application in patients with heart failure poses unique challenges. This is primarily due to overlapping clinical and imaging findings that can make accurate diagnosis difficult.

Tokyo Criteria Overview

The Tokyo Guidelines (TG18) define acute cholecystitis using three diagnostic pillars:

1. **Clinical Symptoms:** Right upper quadrant (RUQ) pain and tenderness, with associated systemic symptoms such as fever or localized peritoneal signs.
2. **Radiologic Findings:** Imaging evidence, including gallbladder wall thickening, pericholecystic fluid, or impacted gallstones.
3. **Inflammatory Response:** Elevated white blood cell counts or biomarkers such as C-reactive protein (CRP) indicating systemic inflammation [1].

These criteria help differentiate acute cholecystitis from other abdominal pathologies. However, pathophysiologic changes in heart failure, particularly right-sided heart failure, mimic these features, creating diagnostic uncertainty.

Challenges in Symptomatology

RUQ Pain in Heart Failure

RUQ pain is a hallmark of acute cholecystitis but is also commonly seen in heart failure, particularly in cases involving significant hepatic venous congestion. The pathophysiologic mechanisms include:

- **Hepatomegaly and Liver Congestion:** Elevated systemic venous pressure in right-sided heart failure

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causes liver enlargement, stretching the liver capsule, and leading to RUQ discomfort or pain.
- **Hepatobiliary Stasis:** Impaired venous drainage may result in bile duct stasis, contributing to pain originating from the liver or gallbladder.
- **Ascites and Peritoneal Stretching:** Chronic fluid accumulation can stretch the peritoneum, causing diffuse abdominal discomfort, often localized to the RUQ.
- **Mesenteric Hypoperfusion:** Reduced intestinal blood flow may lead to ischemia, presenting as cramping or spasm-like pain in the RUQ or other abdominal areas.

Clinical Relevance

In patients with heart failure, RUQ pain is frequently accompanied by systemic signs such as peripheral edema, abdominal fullness, and dyspnea. These symptoms overlap significantly with those of acute cholecystitis, complicating clinical evaluation. Distinguishing between these conditions requires careful assessment of the clinical context, including predisposing cardiac factors [2–4].

Radiological Dilemmas

Gallbladder Wall Thickening in Heart Failure

Gallbladder wall thickening, a key radiologic feature of acute cholecystitis, is also a nonspecific finding frequently observed in heart failure. The underlying mechanisms include:

1. **Hepatic Venous Congestion:** Elevated venous pressure in right-sided heart failure impairs gallbladder drainage, leading to wall edema and thickening.
2. **Hypoperfusion and Hypoxia:** In advanced heart failure, systemic hypoperfusion and hypoxia contribute to inflammation and structural changes in the gallbladder.
3. **Systemic Inflammation:** Elevated levels of acute-phase reactants and cytokines in heart failure exacerbate edema and gallbladder wall thickening.

Clinical Relevance

Gallbladder wall thickening in heart failure is often attributed to hemodynamic changes rather than primary gallbladder pathology. However, distinguishing these changes from acute cholecystitis is challenging. Ultrasound findings should be interpreted with caution, especially when gallstones or pericholecystic fluid are absent. Radiologists and clinicians must collaborate closely to avoid overdiagnosis and unnecessary interventions [5–7].

Inflammatory Response Challenges

Acute Phase Reactants in Heart Failure

Heart failure is associated with elevated levels of acute-phase reactants, complicating the interpretation of systemic inflammatory markers used in the Tokyo Criteria. Two primary mechanisms explain this phenomenon:

1. **Intrinsic Inflammatory Processes:** Some patients with acute heart failure exhibit persistently elevated cytokine levels, such as interleukin-6, suggesting an underlying inflammatory state independent of infection or cholecystitis.
2. **Mesenteric Hypoperfusion:** Reduced intestinal perfusion leads to bacterial or endotoxin translocation, triggering systemic inflammation. Studies have documented elevated endotoxin levels in hepatic veins, correlating with increased acute-phase reactants.

Clinical Relevance

These mechanisms highlight the complexity of interpreting inflammatory markers in heart failure. Elevated CRP or WBC counts may not necessarily indicate infection or acute cholecystitis but could reflect the systemic inflammatory milieu of heart failure. Clinicians should exercise caution when relying solely on these markers for diagnosis [8–10].

The Diagnostic Dilemma

The overlapping features of heart failure and acute cholecystitis—in symptoms, imaging, and inflammatory response—create a significant diagnostic dilemma. Misinterpretation of findings may lead to unnecessary surgical interventions or delays in appropriate treatment.

For example, gallbladder wall thickening observed on ultrasound in a patient with heart failure might be mistakenly attributed to acute cholecystitis, prompting unwarranted cholecystectomy. Conversely, dismissing this finding as a hemodynamic effect of heart failure may delay the recognition of true cholecystitis, increasing patient morbidity and mortality.

Recommendations for Clinical Practice

1. **Multidisciplinary Approach:** Collaboration between cardiologists, surgeons, and radiologists is essential to accurately interpret findings and optimize patient care.
2. **Advanced Imaging Modalities:** Incorporating techniques such as contrast-enhanced ultrasound, magnetic resonance cholangiopancreatography, or hepatobiliary iminodiacetic acid scans may help distinguish between cholecystitis and hemodynamic changes in heart failure.
3. **Dynamic Monitoring:** Serial imaging and inflammatory marker trends can provide additional context, helping differentiate between resolving hemodynamic effects and evolving cholecystitis.
4. **Integrated Scoring Systems:** Developing diagnostic algorithms or scoring systems that account for the

unique interplay between heart failure and cholecystitis may enhance diagnostic accuracy.

Conclusion

Diagnosing acute cholecystitis in patients with heart failure remains a clinical challenge due to substantial overlap in symptoms, imaging findings, and inflammatory responses. The Tokyo Criteria, while invaluable, require cautious application in this patient population to prevent misdiagnosis and inappropriate management.

Multidisciplinary collaboration, advanced imaging, and dynamic monitoring are crucial to navigate this diagnostic intersection. Further research should focus on developing tailored diagnostic tools addressing the unique pathophysiologic overlap between heart failure and acute cholecystitis.

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