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COMPREHENSIVE REVIEW OF CALCULOUS AND ACALCULOUS CHOLECYSTITIS: PATHOPHYSIOLOGY, TREATMENT APPROACHES, AND PREVENTION STRATEGIES

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Abstract: This review offers a detailed academic synthesis of calculous and acalculous cholecystitis, two distinct yet overlapping forms of gallbladder inflammation. It examines their underlying causes, the intricate molecular and cellular processes involved in gallbladder inflammation, contemporary management options (including targeted antimicrobial regimens), and evidence-based preventive measures. Particular attention is given to key mechanisms such as inflammatory signaling pathways, ischemia-reperfusion damage, and the importance of streamlined clinical protocols for optimal patient outcomes.

Introduction

Acute cholecystitis remains a common and clinically significant condition in hepatobiliary medicine. Calculous cholecystitis typically results from physical blockage of the cystic duct by gallstones, whereas acalculous cholecystitis develops without stones, most often in the setting of critical illness or major physiological stress. Both entities share overlapping drivers, including bile stasis, robust inflammatory activation, tissue ischemia, and potential bacterial superinfection.

Etiological Factors

Calculous cholecystitis is primarily linked to the development of cholesterol-based gallstones, which arise from imbalances in bile composition (supersaturation with cholesterol), crystal formation, and impaired gallbladder contractility. Key risk factors include components of metabolic syndrome such as obesity and insulin resistance, female gender, use of estrogen-containing medications, and hereditary influences.

In contrast, acalculous cholecystitis predominantly affects critically ill individuals, including those with major trauma, extensive burns, sepsis, prolonged total parenteral nutrition (TPN), or extended periods of fasting. Its development is multifactorial, centering on gallbladder immobility (stasis), reduced blood flow to the gallbladder wall, and intense systemic inflammatory responses.

Pathogenesis: Molecular and Cellular Insights

In calculous cholecystitis, obstruction of the cystic duct raises pressure inside the gallbladder, impairing mucosal blood supply and leading to ischemic injury. This process activates phospholipase A2, which generates lysolecithin—a substance toxic to the gallbladder lining. The resulting epithelial damage triggers release of pro-inflammatory cytokines (including TNF-α, IL-1β, and IL-6), which in turn activate the NF-κB signaling pathway and amplify the inflammatory response. Oxidative stress through excess reactive oxygen species (ROS) further contributes to mitochondrial impairment. Secondary bacterial invasion, commonly by enteric organisms such as Escherichia coli or Klebsiella species, often worsens tissue injury.

Acalculous cholecystitis is driven mainly by ischemia-reperfusion mechanisms. In critically ill patients, systemic hypotension, endothelial injury, and microvascular clotting lead to hypoxic damage and necrosis of the gallbladder wall. Concentrated, viscous bile combined with

prolonged stasis creates an environment conducive to inflammation, even without mechanical obstruction by stones.

Current Management Strategies

Calculous Cholecystitis

The preferred definitive treatment is early laparoscopic cholecystectomy, recommended within 72 hours of symptom onset in suitable candidates, as supported by randomized evidence showing reduced complications and shorter hospital stays compared with delayed surgery.0d9aca

Antibiotic Therapy: Empirical intravenous antibiotics targeting common biliary pathogens are essential. Common regimens include:

Ceftriaxone (1–2 g IV once daily) combined with metronidazole (500 mg IV every 8 hours)

Piperacillin-tazobactam (4.5 g IV every 6–8 hours)

Amoxicillin-clavulanate (1.2 g IV every 8 hours)

Duration is typically guided by severity and source control; recent guidelines suggest short courses (often 1–4 days post-source control in mild-moderate cases) to support antibiotic stewardship.ea155f

Pain Management: Non-steroidal anti-inflammatory drugs such as ketorolac (30 mg IV every 6 hours) or opioids like morphine (2–4 mg IV as needed) are used for symptom relief.

Acalculous Cholecystitis

Management focuses on supportive care in the intensive care unit, including aggressive fluid resuscitation, hemodynamic support with vasopressors if required, and correction of underlying critical illness. Percutaneous cholecystostomy tube placement serves as an effective bridge or definitive intervention in patients too unstable for surgery.

Antibiotic Therapy: Broader coverage is often warranted due to the severity of illness. Options include carbapenems such as meropenem (1 g IV every 8 hours) or imipenem-cilastatin (500 mg IV every 6 hours), tailored to local resistance patterns and culture results when available.

Preventive Approaches

For Calculous Cholecystitis: Prevention emphasizes lifestyle modification, including weight reduction, dietary control of fats and refined carbohydrates, and management of metabolic risk factors. In selected high-risk patients who are not surgical candidates, ursodeoxycholic acid (UDCA) may help promote bile desaturation and reduce stone formation or related complications.

For Acalculous Cholecystitis: Strategies in at-risk hospitalized or ICU patients include prompt initiation of enteral feeding whenever feasible (to stimulate gallbladder contraction and prevent stasis), careful fluid and hemodynamic management, and close monitoring to avoid prolonged fasting or TPN dependence. Early mobilization and minimization of systemic insults also play important roles.

Conclusion

Effective care of both calculous and acalculous cholecystitis demands a thorough grasp of their mechanical, ischemic, and inflammatory underpinnings. Timely diagnosis, individualized interventions—whether surgical, percutaneous, or pharmacological—and attention to preventive measures are essential for reducing morbidity and improving long-term prognosis in these patients.

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