# Acute Acalculous Cholecystitis in Critically Injured Patients

## *Preoperative Diagnostic Imaging*

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**The potential lethality and predisposing factors of acute acalculous cholecystitis (AAC) are well established; however, preoperative diagnosis remains a challenge. This update of a previous report of 30 cases of AAC at a Level I trauma center describes 14 multiply injured patients who developed AAC and underwent cholecystectomy. All 14 patients had acutely inflamed gallbladders; 6 (42.8%) had areas of necrosis or gangrene. The mortality rate was 7% (1 patient). While the percentage of patients receiving prolonged intensive care (100%), narcotic analgesics (100%), and TPN (93%) correlates with the experience cited previously, the percentage undergoing preoperative diagnostic imaging is unusually high, reflecting a heightened suspicion for AAC. Computed tomographic or sonographic evidence of gallbladder wall thickness ≥4 mm, pericholecystic fluid or subserosal edema without ascites, intramural gas, or a sloughed mucosal membrane was considered diagnostic criteria for AAC. We conclude that preoperative computed tomogram or ultrasound imaging leads to earlier recognition of this life-threatening problem.**

PREDISPOSING FACTORS FOR acute acalculous cholecystitis (AAC) are commonly present in critically injured patients. The mortality rates of up to 75%, described over the past decade, have been attributed to the difficulty in securing a preoperative diagnosis.[1-5]

The diagnostic criteria for AAC on computed tomography (CT) and ultrasound have been previously described at this institution.[6] To assess the impact of a heightened index of suspicion, we undertook a 5-year retrospective study of the radiographic and clinical presentations as well as the clinical outcome of critically injured patients developing AAC.

### Patients and Methods

The records of all patients developing AAC at the Shock Trauma Center of the Maryland Institute for Emergency Medical Services Systems from July 1983 to April 1988 were reviewed. There were ten men and four women. The average age was 36 years (range, 15 to 74 years). Twelve patients were injured in motor vehicle accidents and had multiple-system injuries; one patient was a fall victim; and one was an assault victim with a blow to the head. Patients were evaluated for presence and duration of respiratory failure, length of stay in a critical-care setting, method of alimentation, and mode of analgesia administration. In addition, patients' clinical parameters at the time of diagnosis were reviewed, including CT scan or ultrasound in the 13 patients who had these studies performed, physical findings, laboratory studies, surgical management, pathologic and microbiologic results, and clinical outcome.

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### Results

Table 1 depicts the clinical profile of 14 critically injured patients developing AAC from July 1983 to April 1988. All patients were receiving narcotic analgesia, while most received ventilatory support with PEEP (12) and parenteral nutrition (11). All 14 patients had received prolonged critical care, randomly defined as a stay in an intensive care or neurotrauma critical care unit of seven consecutive days. While all or most patients had fever (14) and leukocytosis (11), abdominal tenderness or liver function test abnormality were present in approximately one half of them.

Of 14 patients with acute acalculous cholecystitis, 13 underwent abdominal CT scanning or sonography in search of intraabdominal sepsis. Eight patients underwent CT scanning alone, three underwent sonography alone,

and two had both studies performed. Major criteria for the diagnosis of gallbladder inflammation include wall thickness ≥4 mm (Figs. 1 and 2), pericholecystic fluid without ascites, intramural gas, and a sloughed mucosal membrane.[6] All 13 patients undergoing preoperative imaging were taken to the operating room with a diagnosis of AAC; surgical findings confirmed the diagnosis. The 14th patient, a 19-year-old man with a severe closed head injury, bilateral pneumothoraces, and a femur fracture from a motor vehicle accident, had developed sepsis and multisystem organ failure by the 36th hospital day. The patient underwent exploratory laparotomy as a desperate measure without preoperative imaging and was found to have an acutely inflamed gallbladder. The patient died on the first day after cholecystectomy.

### Operative Management

All patients were managed with cholecystectomy. All patients had thickened bile or sludge in the gallbladder without gallstones. Pathologic evidence of acute inflammation was present in all cases (Fig. 3). Areas of gangrene were present in 6 cases (Fig. 4).

Operative cultures were positive in four of seven cases in which results were available.

Thirteen of 14 patients survived the operation and were ultimately discharged home or to a rehabilitation center to address their orthopedic or head injuries. This compares favorably with the previously reported mortality rates from this institution (Table 2).[4,5]

TABLE 1. *Clinical Profile of Patients with AAC (MIEMSS, 1983–1988)*

| | N | % |
|---|---|---|
| No. of patients* | 14 | |
| Prolonged critical care | 14 | 100 |
| Fever | 14 | 100 |
| Leukocytosis | 11 | 79 |
| RUQ tenderness | 7 | 50 |
| Hyperbilirubinemia | 7 | 50 |
| ↑ Alk Θ ↑ SGOT | 6 | 43 |
| Narcotic analgesia | 14 | 100 |
| Ventilatory support | 12 | 83 |
| Parenteral nutrition | 11 | 79 |

* Incidence among patients receiving prolonged critical care is 14/2780, or 0.5%.

RUQ, right upper quadrant; Alk Θ, alkaline phosphatase; SGOT, serum glutamic oxaloacetic transaminase.

### Discussion

The potential lethality of acute acalculous cholecystitis has been well established in the old and recent literature.[1–5,7–13] Previously published series are summarized in Table 3,[1,3–5,10,11,14] not as a comparison between these variable patient populations, but to exemplify the seriousness of this affliction, which commonly affects patients already physiologically compromised by the stress of surgery or injury.

Several predisposing factors for the development of AAC have been proposed. Mechanisms by which these factors seem to operate include:

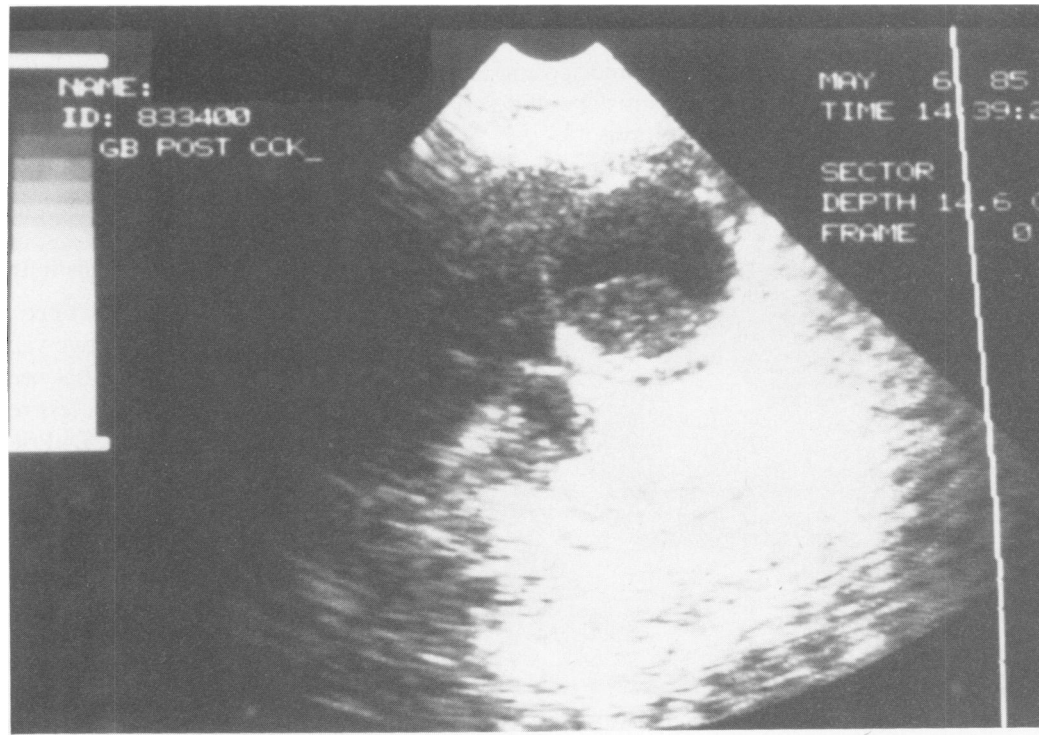


FIG. 1. Ultrasound demonstrating thickened gallbladder wall and sloughed mucosal membrane.

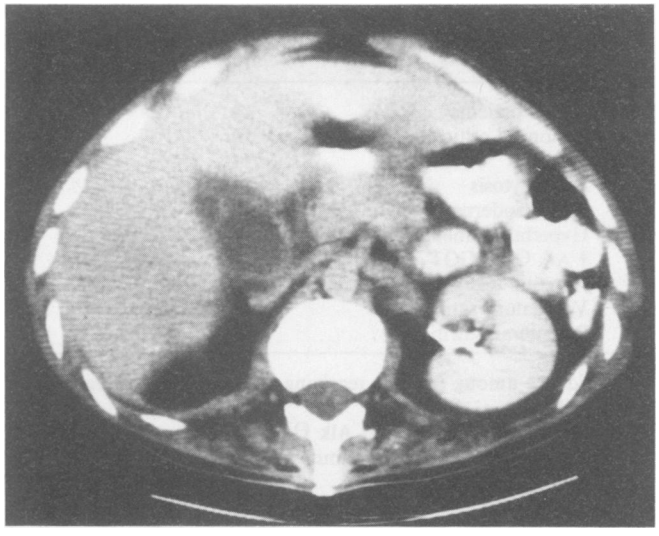

FIG. 2. CT scan demonstrating thickened gallbladder wall.

(1) The relative visceral hypoperfusion seen in the onset and treatment of hypovolemic and septic shock.

(2) Ventilatory support with PEEP, which produces hyperbilirubinemia, decreased portal blood flow, and relative biliary stasis in dogs.[15,16]

(3) Biliary stasis, which occurs from the actions of morphine on the sphincter of Oddi and from the absence of gallbladder contraction in fasting patients.

(4) Finally, gallbladder wall inflammation, which occurs from Hageman factor (XII) dependent pathways. Multiple transfusions, injury, and endotoxemia are among conditions thought to activate these pathways.[1,2]

The frequent occurrence of these predisposing factors in patients with acalculous cholecystitis is well described in series from this and other institutions.[1-5,7-13]

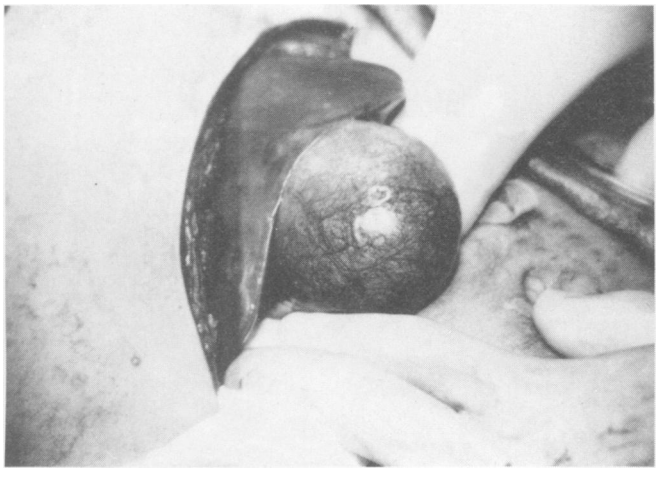

FIG. 3. Acutely inflamed gallbladder (same patient as in Fig. 1).

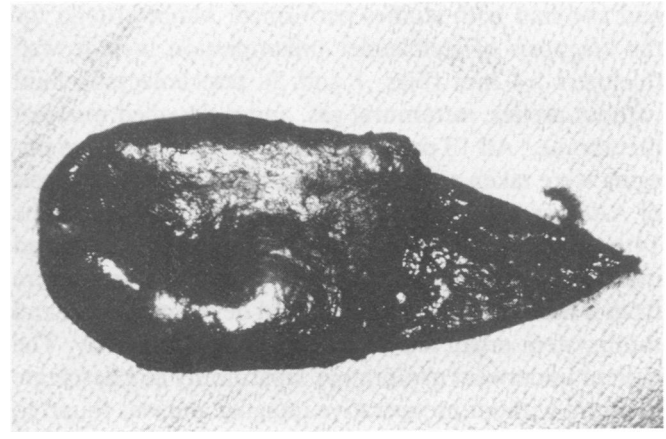

FIG. 4. Gallbladder with sludge and areas of gangrene (same patient as in Fig. 2).

## Diagnosis

The proportion of patients in this report having a diagnosis made before operation (13/14) is unique compared with other series.[3-5,7,10,12,13,17,18] The challenge in securing a preoperative diagnosis arises from the presence of other organ system injuries and the need for analgesia and sedation. Fever and leukocytosis, while commonly present, were too nonspecific to be of great diagnostic benefit and only help to identify a group at risk. Abdominal tenderness, difficult to elicit in this patient population, and liver function abnormalities were absent as frequently as they were present.

During the time period studied, our randomly defined criteria for prolonged stay in a critical care setting (7 days) was met by 2780 patients. Fourteen of these patients (0.5%) developed AAC. The relative infrequence of this disease, even in an institution selectively seeing critically injured patients, further contributes to the challenge of identifying the disease before operation. Thus, the scenario of exploring a patient for a source of sepsis or another diagnosis and finding acalculous cholecystitis still occurs.

## Diagnostic Imaging

During the 5 years described by this study our sense of awareness of the adjunctive value of CT and ultrasound in the diagnosis of AAC has been heightened. This sense is borne of our previous, less rewarding experience with the disease. Specifically, DuPriest et al.[5] and Flancbaum et al.[4] described series of 12 and 18 patients, respectively, who developed acute posttraumatic cholecystitis. Both series were notable for reliance on clinical signs and symptoms (fever, leukocytosis, abdominal tenderness, and sepsis of unknown etiology) for the diagnosis and for limited use of preoperative diagnostic imaging techniques. Preoperative ultrasound examination was performed in 4 of DuPriest's 12 patients; preoperative radionuclide scanning was performed in 1 of Flancbaum's 18 patients.

TABLE 2. *Mortality Rates Among MIEMSS Patients with AAC*

| Span | N | Preoperative Diagnosis | Gangrene | Deaths |
|---|---|---|---|---|
| 1972–1984 | 18 | 1/18 (5.5%) | 12 (66.6%) | 8 (44%) |
| 1983–1988 | 14 | 13/14 (93%) | 6 (42.8%) | 1 (7%) |

Accordingly, the accurate preoperative diagnosis was suspected rarely (2 of 12 and 1 of 18 cases, respectively); gangrene was frequent (7 of 12 and 12 of 18 cases); and mortality was not uncommon (9 of 12 and 8 of 18 cases).

In 1986, Mirvis et al.[6] looked retrospectively at the results of CT and ultrasound in 56 patients evaluated for possible AAC and defined major diagnostic criteria. Specifically, these were (1) gallbladder wall thickness ≥ 4 mm, (2) pericholecystic fluid or subserosal edema without ascites, (3) intramural gas, or a (4) sloughed mucosal membrane. Minor criteria of biliary "sludge" or gallbladder distension were also defined. Parenthetically, they found that percutaneous bile aspiration was of little diagnostic value and that HIDA scanning was compromised by false-positive interpretations in 13 of 24 cases, for a specificity of 38%. Subsequent clinical course (11 cases) or negative surgical exploration (2 cases) contradicted an initial impression of acute acalculous cholecystitis. The limited value of HIDA scanning in this patient population corroborates the experience of other authors.[19,20] In contrast, there were no false-positive CTs (100% specificity) and one false-positive ultrasound (96% specificity), leading to our emphasis on these modalities as diagnostic adjuncts.

Selection of CT *versus* ultrasound was based on patients' clinical status. CT scanning has the advantage of detecting other foci of intraabdominal sepsis. Ultrasound is less expensive and can be performed at the patient's bedside. Two of our 13 patients had equivocal CT scans; ultrasound was performed, confirming the diagnosis of AAC. Eight patients had CT scans alone, while three had ultrasounds alone.

## Treatment and Results

Our experience concurs with that of others who advocate that cholecystectomy should be performed whenever possible for acalculous cholecystitis.[4,5,7–10,12,17,18,21] Cholecystectomy was performed on all 14 of our patients. We believe that the improved rate of mortality over the last 5 years is due in part to a higher index of suspicion leading to more common use of diagnostic imaging adjuncts in clinically appropriate patients. The need for early diagnosis and treatment of AAC is well established.

We conclude that a heightened index of suspicion and adjunctive use of CT or ultrasound are important modalities in the prompt diagnosis and treatment of acute acalculous cholecystitis in critically injured patients.

TABLE 3. *Mortality Rates Associated with AAC*

| Series | N | Mortality Rate (%) |
|---|---|---|
| Lindberg et al.; 1970 | 12 | 16.6 |
| Glenn and Becker; 1979 | 139 | 6.5 |
| Howard; 1981 | 63 | 17.6 |
| DuPriest et al.; 1979 | 12 | 75 |
| DuPriest et al. (literature review); 1979 | 98 | 33 |
| Long et al.; 1978 | 17 | 36 |
| Herlin et al.; 1982 | 11 | 18 |
| Flancbaum et al.; 1985 | 18 | 44.4 |

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