MANAGEMENT OF ACUTE CHOLECYSTITIS

Analysis of 67 Consecutive Cases

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General Considerations

ACUTE cholecystitis is essentially a surgical problem. Nearly all cases begin as the result of the mechanical action of gallstones, either by obstruction of the cystic duct or by erosive action on the wall of the gallbladder. Bacterial invasion almost invariably is secondary to the mechanical factor and may occur a matter of days after the onset of symptoms, or not at all. Removal of the gallbladder and its contained stones effects a cure.

It would seem that once the disease has begun and the diagnosis has been established, the therapeutic aim should be prompt cholecystectomy. Yet case analyses show that delay in surgery is the rule rather than the exception. Further, many internists and family physicians and some surgeons record their belief that operations should not be performed during an acute phase of the disease, and that symptoms should be permitted to subside completely; elective cholecystectomy is then to be performed during a quiescent interval some weeks or months thereafter. It is our belief that in most instances substantial delay is not justified and may result in needless suffering, prolonged loss of the patient's time from productive activity and, occasionally, in the development of otherwise avoidable complications.

What are the arguments advanced by those who favor procrastination?

1. "Most attacks of gallbladder pain subside spontaneously and promptly."

It is true that the average attack of gallbladder colic—almost always the precursor of acute cholecystitis—will last only a few minutes or an hour or two, or will disappear after the administration of a narcotic. It is also true that the borderline between "colic" and "acute cholecystitis" is a hazy one. Perhaps the clinician's thinking would be clarified by the establishment of some arbitrary definitions to aid in the approach to therapy. If pain has not completely disappeared within six hours of onset, the clinician should assume that he is confronted with acute cholecystitis and he should regard his patient as a candidate for urgent operation. The same may be said if symptoms recur a

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few hours after they have been relieved. Though attacks of gallbladder colic commonly subside promptly, there are enough exceptions to this rule to reduce its clinical effectiveness.

2. "The diagnosis is not always clear, and nonsurgical disease may be producing the symptoms."

Often previous roentgen studies will have shown the presence of gallstones, or the gallbladder will have failed to be visualized with the "double-dose" technic. If such a patient should develop mid-epigastric or right upper quadrant pain, tenderness over the region of the gallbladder, and constitutional evidences of an inflammatory process such as fever, tachycardia, or leukocytosis, acute cholecystitis may be presumed without further diagnostic study. However, when these signs and symptoms are present, but there never has been roentgen proof of gallstones or nonfilling of the gallbladder, the problem is different. Here, further diagnostic procedures are in order to avoid a possibly useless operation. The first step is to obtain a plain film of the abdomen; in 10 per cent of patients with gallstones there is sufficient calcium in the stones to cast a characteristic shadow without the use of contrast medium. The evidence of stones will confirm the diagnosis and justify prompt operation.

If the "scout film" does not show stones, diagnostic delay to permit cholecystographic studies is fully justified provided that the patient's general condition permits. It is our practice to give such a patient a double dose of oral contrast medium (to avoid the necessity of repeating the test the following day) and, if clinical suspicion runs high, tentatively to schedule the patient for operation immediately after the interpretation of the cholecystogram. If stones are demonstrated or the gallbladder fails to be visualized, operation is performed. If the roentgen findings show the gallbladder to be normal, surgery is canceled and further observation with additional diagnostic studies is undertaken.

For the patients who are unable to take the oral contrast medium or who will vomit the pills, intravenous cholecystographic studies may be substituted, with suitable allowance of time for the gallbladder to fill. If the intravenous contrast medium cannot be given because of drug sensitivity, the patient's general condition must then be the sole guide to therapy.

It has been argued that cholecystographic contrast media taken orally may actually precipitate an attack of acute cholecystitis, or aggravate an existing one. The evidence supporting this argument is scant, and the counterposition may be taken that even if aggravation of symptoms does occur, this will clarify the diagnosis, and in any event is taking place under careful scrutiny, in the hospital, where it could hardly result in any material harm to the patient.

3. "Technical difficulties are increased when surgery is attempted on the so-called 'hot' gallbladder, and, by implication, the chances for a surgical miscue."

With modern operating room facilities the well-trained surgeon is fully able to cope with the increased technical difficulty presented by an acutely inflamed gallbladder. The operation may be characterized as one that is "hard on the surgeon but easy on the patient."

In the first place, the surgeon has always available the expedient of performing a cholecystostomy: emptying the gallbladder of stones and inserting
a tube for decompression. This will invariably relieve the acute attack. Cholecystostomy normally is succeeded three or four months later by a secondary cholecystectomy, since otherwise re-formation of stones and more attacks will follow. Cholecystostomy will not be necessary in the great majority of patients, but in the extremely ill it may be a lifesaving measure that can be carried out with minimal anesthesia; it may also be the wisest course for the surgeon to follow in patients in whom the usual landmarks have been totally obscured by local inflammation.

In the average patient, however, cholecystectomy will be perfectly feasible. It usually is best to decompress the gallbladder first so that it may be grasped more readily by instruments, and it nearly always is wisest to remove it from the fundus down, performing the dissection millimeter by millimeter until the cystic vessels and the cystic duct are encountered and are ligated. Bleeding from the gallbladder fossa nearly always will subside with light packing and pressure from a malleable retractor, although it is prudent to be prepared for a possible blood transfusion.

In most instances it will be possible to obtain an operative cholangiogram; this should be made since a small proportion of patients with acute cholecystitis will also have stones in the common bile duct (5 per cent in our series). In some patients the inflammatory process will be so severe that cholangiography will be difficult, and exploration of the common bile duct unwise. The surgeon must always exercise caution to avoid excessive pressure while injecting the contrast medium into the common bile duct, lest infection present in the bile ducts be disseminated further. On more than one occasion we have seen a gangrenous gallbladder "come off" in the surgeon's hand without either recognition or ligation of the cystic vessels or the cystic duct; there evidently had been thrombosis and the duct had been obliterated. In such cases it is highly unlikely that stones are present in the common bile duct, and it is better for the patient if the surgeon contents himself with a simple removal of the gallbladder.

Although the surgery of acute cholecystitis is usually more difficult than routine gallbladder operations technically, if the surgeon observes the precautions noted and, above all, is willing to perform an occasional cholecystostomy, no additional danger should accrue to the patient; and if the operation does present technical problems, experience has shown that it takes months for the inflammation to subside sufficiently to make an interval operation much simpler. During this waiting period, on the assumption that operation should be performed only when the disease is in a quiescent phase, another attack is more than likely to supervene, raising the same problems that arose with the first one.

Analysis of Cases

In reviewing cases to see whether they fulfill diagnostic criteria for acute cholecystitis, certain difficulties become apparent. The histopathology of the
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removed gallbladder presents problems unlike those in acute appendicitis. Commonly, the gallbladder has had repeated bouts of inflammation, and an acute attack is superimposed on chronic changes in the wall. It is not an uncommon experience for the surgeon to find a tensely distended gallbladder, with a thickened wall, containing “white bile,” the so-called “hydrops,” in a patient who is essentially asymptomatic at the time of operation. Grossly, such a gallbladder may be indistinguishable from the acutely inflamed gallbladder in a patient experiencing severe symptoms. The microscopic pattern may verify the clinical impression that the condition is indeed chronic. The converse, however, may not be the case; occasionally, such a gallbladder in a patient who has pain and fever, and tenderness over the gallbladder area, also will show no acute inflammation histologically. Hence, the microscopic diagnosis of acute cholecystitis cannot be regarded as the final, arbitrary factor in establishing the diagnosis of the acute episode. It is best to depend on a correlation of the clinical picture presented by the patient, and the gross findings at operation as well as the microscopic picture.

In a consecutive personal series (S. O. H.) of 363 cholecystectomies (excluding incidental cholecystectomy performed in the course of another operation, such as gastric resection for ulcer), 100 cases were regarded as possibly qualifying for study as acute cholecystitis. This number was reduced by eliminating 28 cases in which there was no record of recent pain or the pain was present longer than a week prior to admission to the hospital. All of these 28 patients had gallstones, and in eight the pathologic diagnosis was “chronic recurrent acute cholecystitis,” qualifying them from histologic evidence as “acute;” in two patients the pathologic diagnosis was hydrops. Six of the eight who showed acute inflammation in the removed gallbladder had had symptoms from 8 to 29 days, and perhaps could be regarded clinically as representing a “subacute” phase of the disease. Five additional cases were excluded either because both pain and tenderness were absent (three cases) or the pain or tenderness was so located that the gallbladder could hardly have been responsible; one patient of this group showed acute inflammation microscopically. Since all of these patients survived postoperatively their exclusion does not affect the over-all findings or conclusions.

The 67 patients classified as having acute cholecystitis all had the onset of upper abdominal pain a week or less prior to hospitalization, and in most of them the pain was localized to the right upper quadrant or the epigastrium; all but three showed appropriate abdominal tenderness at the time of admission. (In an occasional obese patient with a deeply placed gallbladder protected by an overhanging liver, local tenderness may be absent; in most cases, however, fist percussion over the lower ribs alternately on both sides will elicit a characteristic difference in sensation between right and left, the jar being noticeable to the patient on his right side even though the discomfort is not severe.)

Forty patients had at least a degree of fever, and only 12 had a normal temperature. Leukocytosis seemed to be correlated to the presence of fever, so that only two of the patients with normal temperature had a leukocyte count greater than 10,000 per cu. mm. On the other hand, the microscopic findings
included acute inflammatory changes in all of the 12 patients with normal temperature, confirming the clinical and operative impressions.

Of the 67 patients there were 39 men and 29 women. This contrasts with a ratio of 3 women to 1 man in the remainder of this series. Perhaps the apparent discrepancy may be explained on the basis that men are more likely to refuse cholecystectomy for the mere presence of stones and an occasional short bout of colic, and a serious complication is necessary before they leave their jobs to enter the hospital. The age span in this group ranged from 25 to 75 years, both extremes represented by women.

Pathologic Findings

In all but three patients gallstones were present. In these three the pathologic report was “chronic recurrent acute cholecystitis”; in two there was a positive culture, and in the third there was a coexisting (and possibly causative) acute pancreatitis. One of the two patients with a positive bile culture had a cyst of the common bile duct, presumably congenital. Three of the 64 patients having stones in the gallbladder had stones in the common bile duct as well, although the incidence is considerably lower for this group than in the elective group (where in our experience it is more than 20 per cent). Nevertheless, it would seem that cholangiography is warranted, when it can be safely and conveniently done, to avoid secondary operations. Furthermore, if the gallbladder is acute cholecystitis should be stoneless, “reflux” cholecystitis as a result of common bile duct obstruction from a stone, or pancreatitis with inflammation resulting from the reflux of pancreatic juice into the gallbladder, should be looked for via cholangiography. Although a single large stone can produce an “erosive” cholecystitis and it takes only a single small stone to obstruct a cystic duct, 50 of the 64 patients with stones had multiple stones.

The histopathologic diagnoses listed by the pathologist were as follows (biopsies were performed in those patients having cholecystostomy):

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Number of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic recurrent acute cholecystitis</td>
<td>43</td>
</tr>
<tr>
<td>Acute cholecystitis—</td>
<td>16</td>
</tr>
<tr>
<td>(with gangrene 4)</td>
<td></td>
</tr>
<tr>
<td>(with perforation 1)</td>
<td></td>
</tr>
<tr>
<td>Granulation tissue (abscess)</td>
<td>1</td>
</tr>
<tr>
<td>Chronic cholecystitis—</td>
<td>7</td>
</tr>
<tr>
<td>(with hydrops 1)</td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>67</strong></td>
</tr>
</tbody>
</table>

As previously noted, the absence of acute inflammation microscopically does not negate the clinical diagnosis of acute disease. In at least one patient
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The clinical picture was that of a continuing colic without actually producing the total obstruction usually seen.

Cultures of the bile were made in 55 of the 67 patients. The culture was sterile in 34 instances, confirming the well-known belief that in its initial stages the inflammation is of mechanical, not of bacterial, origin. The commonest organisms cultured were, Escherichia coli (seven patients), Staphylococcus albus (seven patients), and nonhemolytic streptococci (four patients). A higher incidence of positive cultures might have resulted if the wall of the gallbladder rather than the bile had been cultured.

Treatment and Results

Of the 67 patients, 61 were treated by primary cholecystectomy; one patient died in the hospital.

**Case 1.** A 70-year-old white woman, was admitted to a medical service here because of progressive difficulty in walking, pains in the legs, and gallstones. There was a marked language barrier. She was seen early in the hospital course by the surgical consultant because of an attack of abdominal pain, and right upper quadrant tenderness seemed to be present. Nonsurgical treatment of the symptomatic gallstones was urged in view of the uncertain nature of the basic disorder, presumed to be neurologic. Various studies did not clarify the diagnosis, and since the patient continued to have attacks of severe pain, operation was carried out two weeks after the patient was first admitted. At operation the gallbladder was seen to be large, tense, and thick-walled; in its distal portion a large calculus obstructed the cystic duct. Cholecystectomy presented no special difficulty and operative cholangiograms showed a normal common bile duct. Pathologic diagnosis was chronic cholecystitis.

Postoperatively, the patient did not do well because of progressive generalized weakness and pulmonary complications. She could not cough effectively and ultimately bronchopneumonia and atelectasis developed which failed to respond. Repeated bronchoscopies were carried out in an effort to clear the respiratory passages, but she died 12 days after the operation, and 25 days after hospital admission. Postmortem examination revealed for the first time that the basic neurologic disorder was amyotrophic lateral sclerosis and the progressive weakness led to the fatal outcome. Although judgment could be criticized for operating on this patient, she did present a continuing problem in biliary tract pain, and death, although doubtless hastened by surgery, was due primarily to other causes.

Of the 61 patients having cholecystectomy, cholangiograms were made in 46, and 11 of these underwent an exploration of the common bile duct in addition; stones in the common bile duct were present in two. (The third patient having a stone in the common bile duct underwent primary cholecystostomy). Cholecystostomy was performed in six patients. One of these patients died shortly after readmission 16 days after his operation and three days after his initial discharge.

**Case 2.** A 65-year-old white man was admitted as an emergency at 4 a.m. with severe epigastric pain that began four days previously and was associated with vomiting. On
physical examination there was a rectal temperature of 101°F., a pulse rate of 96, and a
tender mass in the right upper quadrant. An electrocardiogram showed atrial fibrilla-
tion. Despite supportive measures with gastrointestinal siphonage, intravenous fluids,
and antibiotics, and including digitalization, his condition worsened and, approxi-
mately 10 hours after admission to the hospital, a cholecystostomy was performed under
intercostal procaine hydrochloride block. The gallbladder was tense and the dome
was a mottled, purplish red. When the gallbladder was opened, thin, bloody fluid first
escaped, followed by dark, thick bile. The mucosal lining of the gallbladder was shaggy,
but no stones were found. A biopsy showed acute cholecystitis, in part necrotizing. A
catheter was placed in the gallbladder and the abdomen was closed. A culture of the
bile was sterile.

The postoperative course was uneventful except that cholecystocholangiograms
demonstrated a calculus impacted in the cystic duct. The patient ran a low-grade fever
for a few days but had been normal for 48 hours at the time of his discharge on the
thirteenth postoperative day.

Three days later, 16 days after the cholecystostomy, he was readmitted moribund,
with no blood pressure or pulse obtainable. A few hours earlier he had severe substernal
pain. Despite all efforts at support he died several hours after admission. A postmortem
examination showed that death was due to massive thrombosis of portal, splenic, superior
and inferior mesenteric veins, infarction of proximal jejunum, and necrosis of the right
lobe of the liver. It was believed that this was a coincidental fatal disease occurring
after an otherwise satisfactory convalescence. Although the calculus had caused the
acute cholecystitis and was not removed at operation, it did not seem to be in any way
related to the massive mesenteric thrombosis.

The principal indication for cholecystostomy in these patients was the
coexistence of other constitutional infirmity or disease that rendered a prompt
relief of symptoms with minimal anesthesia desirable. We believe that a planned
cholecystostomy is always justifiable in poor-risk patients; we also believe that
it may be resorted to without hesitation or apology whenever in the surgeon's
opinion local operative conditions, such as intense inflammation, poor exposure,
or a poorly taken anesthetic, render it desirable.

"Early" Versus "Late" Operation

The physician in most instances cannot control the time in the course of the
disease when he first sees the patient, and hence often cannot recommend or
perform an "early" operation. Table 1 indicates the duration of symptoms at
the time the patients entered the hospital, and the elapsed time between onset
of symptoms and operation. "Early" operation is clearly impossible for those
patients who enter the hospital more than two days after the onset of symptoms,
but it is still possible for the physician and the surgeon to reduce the hospital
stay by avoiding further unnecessary delay.

Table 2 shows that hospital delay may seem excessive even when the policy
is to operate promptly. Twenty-five per cent of the patients in this series were
operated upon more than five days after entering the hospital.
Table 1.—Correlation of duration of symptoms from time of onset to hospitalization and to operation for acute cholecystitis in 67 patients

<table>
<thead>
<tr>
<th>Total time from onset of symptoms to hospitalization</th>
<th>Total time from onset of symptoms to operation, number of patients</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>24 hours or less</td>
</tr>
<tr>
<td>24 hours or less</td>
<td>27</td>
</tr>
<tr>
<td>2 days</td>
<td>9</td>
</tr>
<tr>
<td>3 days</td>
<td>9</td>
</tr>
<tr>
<td>4 days</td>
<td>5</td>
</tr>
<tr>
<td>5 to 7 days</td>
<td>17</td>
</tr>
<tr>
<td>Grand Total</td>
<td>67</td>
</tr>
</tbody>
</table>

Table 2.—Delay in hospital—elapsed time from hospital admission to operation, cumulative figures

<table>
<thead>
<tr>
<th>Hospital delay</th>
<th>Number of cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>More than—</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 days</td>
<td>16</td>
<td>24%</td>
</tr>
<tr>
<td>4 days</td>
<td>21</td>
<td>31%</td>
</tr>
<tr>
<td>3 days</td>
<td>25</td>
<td>37%</td>
</tr>
<tr>
<td>2 days</td>
<td>34</td>
<td>51%</td>
</tr>
<tr>
<td>1 day</td>
<td>56</td>
<td>84%</td>
</tr>
<tr>
<td>Within one day</td>
<td>11</td>
<td>16%</td>
</tr>
</tbody>
</table>

Summary and Conclusions

Acute cholecystitis in most instances is caused by the mechanical action of gallstones and is curable by cholecystectomy. The initiating factor is obstruction of the cystic duct, or mucosal erosion, by the gallstones. Bacterial invasion is secondary and late. Prompt surgical intervention is indicated once the diagnosis is established. In the sick or elderly patient, or in the patient in whom inflammatory changes render the operation unusually difficult, the surgeon should not hesitate to perform a cholecystostomy.

Data from series of 67 surgical patients having acute cholecystitis are reviewed. There were two deaths but, after complete postmortem study, they did not seem to be directly related either to the disease or to the operative procedure.