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Acute Acalculous Cholecystitis in a Severely Burned Patient
— A Case Report —

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ABSTRACT. A 56-year-old woman, suffering from major third degree burns, had right hypochondric pain and fever on her 57th post-burn day. A diagnosis of acute acalculous cholecystitis was established by ultrasonography. Despite effective drainage of the gallbladder through PTGBD, the patient died of ARDS and septic shock on the 73rd post-burn day.

Acute acalculous cholecystitis is an uncommon but potentially lethal complication in severely burned patients. Ultrasonography is helpful in early diagnosis and drainage. PTGBD is indicated in severely burned patients.

Key words : acalculous cholecystitis — burn — PTGBD

Acalculous cholecystitis accounts for 2-14 percent of all cases of acute cholecystitis. Although the incidence is higher in posttraumatic, postoperative and pediatric patients, severely burned patients are also at risk for this condition.

In this case study, we report a case of acute acalculous cholecystitis in a severely burned patient.

CASE REPORT

A 56-year-old woman sustained flame burns to 40 percent of her total body surface area (TBSA). The burns were mainly on the face, neck, chest wall and upper back with 30 percent being third degree burns.

The patient was resuscitated using Purkland formula, and a tracheostomy was carried out. Sequential excision and grafting was performed on the 7th post-burn day. A total volume of 3,400 ml blood was transfused and total parenteral nutrition (TPN) was maintained. Oral meal intake was started on the 32nd post-burn day.

The patient showed a good clinical course until the 57th post-burn day. The raw surface grew smaller, but the burn wound cultures revealed *Staphylococcus aureus* and *Pseudomonas aeruginosa.*

On the 57th day, a temperature of 40.1°C, pain and tenderness in the right upper quadrant were recorded. Laboratory test results revealed the following : hematocrit at 23.6 percent, platelet count at 156,000, and white-cell count at 15,900, with 22 percent band forms. Bilirubin was 1.1 mg per 100 ml, alkaline phosphatase (ALP) was 269 U, lactic dehydrogenase (LDH) 177 U,

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glutamate oxaloacetate transaminase (GOT) 104 U, glutamate pyruvate transaminase (GPT) 131 U, gamma glutamyl transpeptidase (γ GTP) 131 U, amylase 140 U per liter, and serum osmolarity was at 295 mOsm per kilogram. Administration of Cefperazone was begun.

On the 59th post-burn day, ultrasonography revealed a thickened, dilated gallbladder measuring approximately $15 \times 5 \times 5$ cm, containing sludge (Fig. 1). Percutaneous transhepatic gallbladder drainage (PTGBD) was performed immediately. Ultrasonography and cholecystography from the drainage tube showed no gallstones (Fig. 2). Bile and blood cultures grew *Staphylococcus aureus.*

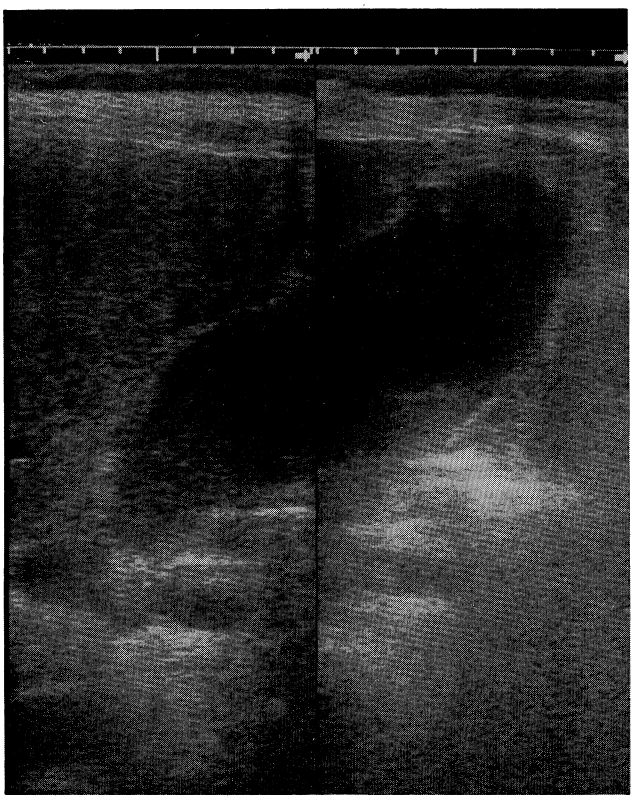

Fig. 1. Ultrasonography on the 59th post-burn day.
Ultrasonography revealed a thickened, dilated gallbladder measuring approximately $15 \times 5 \times 5$ cm, containing sludge.

Following PTGBD, the fever decreased and abdominal pains subsided. Ultrasonography showed no dilatation of the gallbladder. However, respiratory failure worsened and sepsis became critical. The patient expired on the 73rd post-burn day. An autopsy was performed.

Pathological Findings: The gallbladder was extremely small, necrotic and covered with great omentum. There were no stones in the gallbladder or bile duct.

Microscopic examination of the gallbladder revealed necrotic changes with light polymorphonuclear leukocytic infiltration throughout the wall of the gallbladder (Fig. 3).

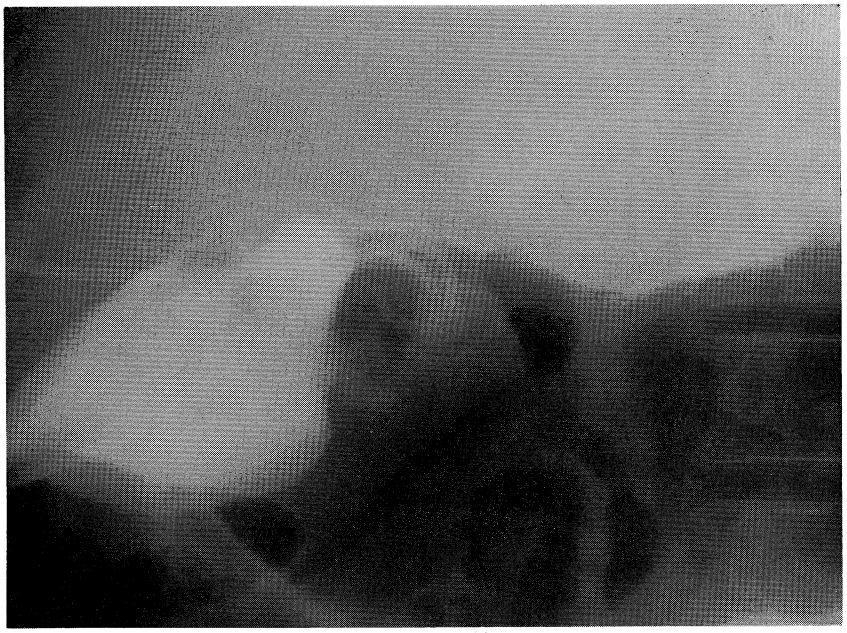

Fig. 2. Cholecystography from the drainage tube. There were no stones in the gallbladder.

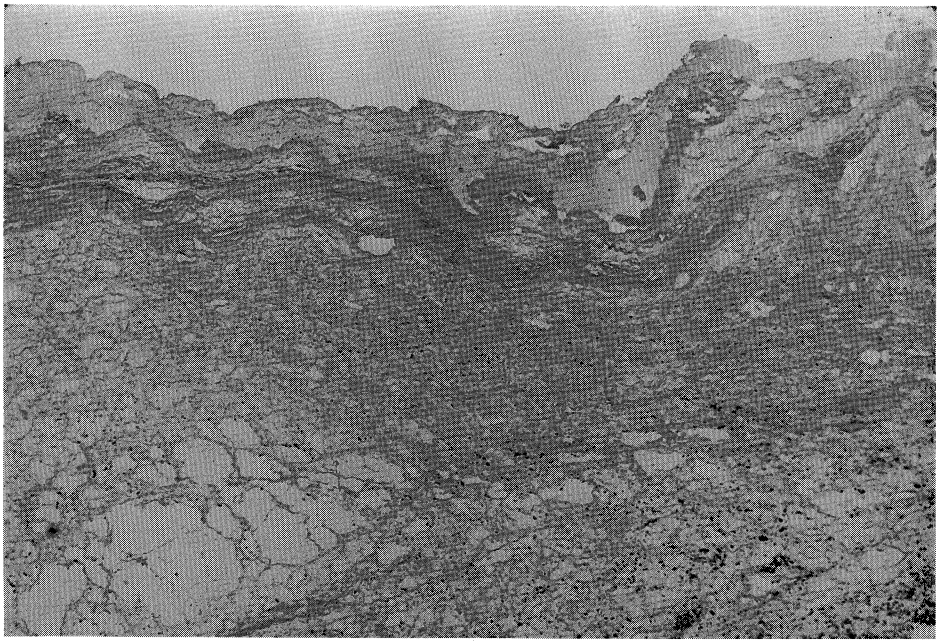

Fig. 3. Microscopic examination of the gallbladder.
Microscopic examination revealed necrotic changes with light leukocytic infiltration throughout the wall of gallbladder.

DISCUSSION

Acalculous cholecystitis accounts for 2–14 percent of all cases of acute cholecystitis.[1,2] The incidence is higher in pediatric,[3] posttraumatic,[4] postoperative

TABLE 1. Summary of cases of acalculous cholecystitis following burn injury since 1970

		Age	Sex	BSA	Onset (Post-burn day)	Therapy	Outcome	Septicemia
Munster	1971	22	M		15		+	(−)
〃		20	M		42		+	Staphylococcus, Candida
〃		20	M		14		+	Pseudomonas
〃		25	M		12		+	Staphylococcus
〃		17	M	mean	9		+	Pseudomonas, Aerobacter
〃		41	M	56.2%	9		+	Pseudomonas
〃		61	M		8		+	(−)
〃		48	M		9		+	Staphylococcus, Aerobacter
〃		20	M		12	Cholecystectomy	○	Klebsiella, Aerobacter
〃		20	M		10	〃	○	(−)
Ternberg	1975	2	F	—	4	Cholecystostomy	+	Pseudomonas
Alawneh	1978	33	M	48	22	Cholecystectomy	○	(−)
〃		40	M	60	18	〃	○	Candida
〃		56	M	62	37	〃	+	Staphylococcus
〃		28	F	38	16	〃	○	Staphylococcus
〃		34	M	54	25	〃	○	Pseudomonas
〃		40	M	35	10	〃	○	Klebsiella
〃		69	M	28	34	〃	○	Candida
Chen	1978	55	M	50	6	〃	○	Staphylococcus
Glenn	1979	36	M	—	14	〃	○	
Rice	1980	26	M	55	31	〃	○	(−)
〃		29	M	45	35	〃	○	
Glenn	1982	65	M	50	17	〃	+	
〃		4	F	33	14		+	
〃		58	M	33	13		+	
Gately	1983	19	M	50		Cholecystectomy	○	
McDermott	1985	32	M	40	30	〃	○	Staphylococcus, Proteus
〃		22	M	37	23	Cholecystostomy	○	(−)
〃		23	M	75	16	〃	○	(−)
〃		40	M	50	62	Cholecystectomy	○	Klebsiella, E. coli, Candida
Ross	1987	26	M	41	33	〃	○	Staphylococcus, Enterobacter
〃		58	M	60	27	PTGBD	○	(−)
〃		33	F	22	19	Conservative therapy	○	Serratia
Present report		56	F	40	56	PTGBD	+	Staphylococcus

○ : survived + : died

and burn patients.[2,5,6] Recent articles have reported an overall incidence rate of 0.4 to 0.5 percent for acute acalculous cholecystitis in burn patients.[2,6] The frequency is higher in men and among younger populations. It is especially high when the burn surface area is greater than 30 percent of the TBSA (Table 1).[1–10]

In Japan, however, only one case has so far been reported.[11] This present case may be the second report of acute acalculous cholecystitis in Japan.

Various hypotheses have been proposed as to the cause of acalculous cholecystitis including bile stasis, sepsis and ischemia. Glenn and Becker emphasized the activation of factor XII-dependent pathways in causing acalculous cholecystitis.[1] TPN is also known to cause acalculous cholecystitis due to bile stasis. Multiple blood transfusions have also been was proposed as another cause because increased bilirubin loads into the gallbladder.[12]

In this case, however, neither bile stasis nor ischemia were the cause of the acute cholecystitis because oral meal intake was immediately begun and vital signs were stable. Bilirubin was within the normal range at the time of onset. In this case, sepsis due to the growth of *staphylococcus aureus* in the blood and bile was the cause. Such growth has also been noted in many other reported cases.

Frequent symptoms of acute acalculous cholecystitis are right upper quadrant pain or tenderness, fever and leukocytosis. But diagnosis is usually complicated due to local injury or endotracheal intubation. Ultrasonography is helpful in early diagnosis of acute cholecystitis with findings of a dilated gallbladder with sludge and wall thickening.[7,13] This procedure can be done at the bedside in the intensive care unit. Ultrasonography is also helpful when performing PTGBD.

Early recognition and surgical intervention are the main forms of treatment for acute cholecystitis.[1] Cholecystectomy is the first choice because of the high proportion of cases with gangrene or perforation and because it is normally performed with good results.[2,3] Recently, PTGBD has been advocated in critically ill patients. In this case, the clinical symptoms and signs subsided after PTGBD and little evidence of cholecystitis was found at autopsy. Therefore, PTGBD seemed effective.

The morbidity and mortality for patients with acute acalculous cholecystitis is much higher than for patients with calculous disease.[1] In a recent study, McDermott and his associates reviewed 27 cases of acute acalculous cholecystitis in severely burned patients from 1960 and reported an overall death rate of 51.8 percent.[5]

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