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BILIARY PERITONITIS AND ACUTE STRESS-INDUCED CHOLECYSTITIS IN A SEVERE BURN PATIENT: RADIOLOGICAL AND ANESTHETIC FEATURES

A case report and review of the literature

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Abstract: Acute stress-induced cholecystitisis an inflammation of the gallbladder that most often occurs in the intensive care setting. It may be lithiasis or alithiasis and is complicated by perforation in 10% of cases. A positive diagnosis is difficult and relies on a range of clinical, biological, and radiological factors, some of which may be non-specific. The Niemeier classification defines three types of perforation and helps guide management. We report a case of biliary peritonitis complicating alithiasic cholecystitis in a setting of severe skin burns, documented in our hospital facility by reviewing data from the literature.

Index Terms **- biliary peritonitis, alithiasic cholecystitis, cholecystectomy, extensive burns, CT scan.**

I. Introduction:

Cholecystitis corresponds to inflammation of the gallbladder wall, often of lithiasic origin. The alithiasic form is rarer, occurring mainly in intensive care units [4].

Vesicular perforation is a serious and potentially fatal complication of cholecystitis [2]; Niemeier's classification describes three types of vesicular perforation, from which a diagnostic and therapeutic strategy is derived.

II. Case report:

We report the case of a 16-year-old young man, with no pathological history, admitted to intensive care for grade 2 burns extending to 40% of his body surface, who reported generalized abdominal pain evolving for 1 day with a starting point in the right hypochondrium.

Clinical examination revealed tachycardia at 115 bpm, hypotension at 100/60 mmHg, and polypnea at 30 cycles/min. Biological workup showed hyperleukocytosis of 15,000/mm3, predominantly neutrophils, and elevated CRP of 125 mg/L, with no disturbance of liver function.

Ultrasound was limited by skin burns, and the patient underwent an abdominal CT scan with and without iodinated contrast. The latter revealed a medium-volume peritoneal effusion in the perihepatic area, in the right parietocolic gutter, and in the pouch of Douglas. The gallbladder was semifull, with thickening and a fundial parietal contrast defect, with a small collection opposite "**Fig. 1**". There was no evidence of intravesicular lithiasis.

The diagnosis of biliary peritonitis due to alithiasic cholecystitis was confirmed during an exploratory laparotomy, which revealed a bilious and hematic peritoneal effusion and a perforation of the vesicular fundus, sealed by a small collection "**Fig. 2**".

The patient underwent retrograde cholecystectomy and peritoneal lavage under general anesthesia: Intubation by video-laryngoscope in rapid sequence with massive vascular filling was performed with crystalloids through a central venous catheter and placed on a 1 mg/hour noradrenaline continuous infusion with cardiovascular monitoring by an arterial catheter. Induction was done with 80 mg of ketamine and 50 mg of rocuronium. 300 gamma of fentanyl was administrated for analgesia.

Anesthesia was maintained with sevoflurane at a minimum alveolar concentration of 1.5%. The depth of anesthesia was monitored using a bispectral index. During the procedure, the patient experienced two episodes of hypotension, which were managed by increasing the dose of noradrenaline. Antibiotic prophylaxis was initiated with 2 g of amoxicillin-clavulanic acid, 500 mg of metronidazole, and 160 mg of gentamycin. Post-operatively, the patient was extubated without incident, and the post-operative course was simple. He was referred to the plastic surgery department for further treatment.

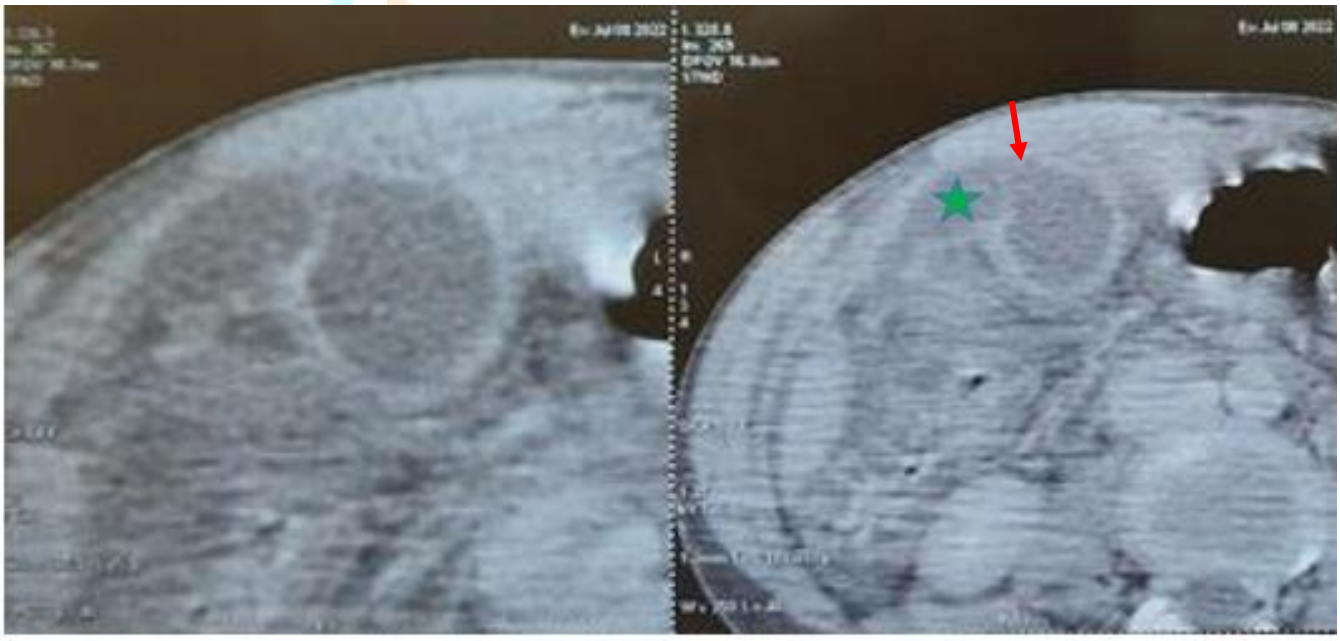

Figure 1: Abdominal CT scan in axial section with injection of iodinated PDC: note the contrast defect in the vesicular wall ( ) and the peri-vesicular collection ( ).

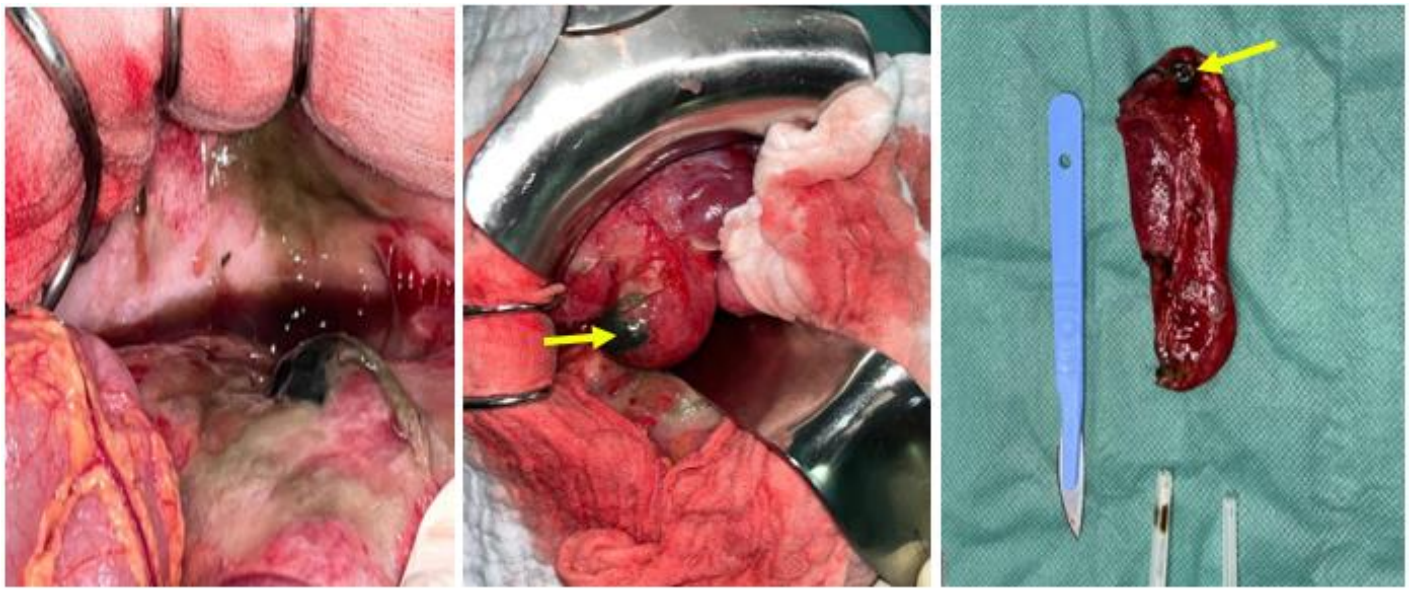

Figure 2 : per operative images of retrograde cholecystectomy: note the colematte fundic perforation ( ) and peritoneal effusion.

III. Discussion:

Stress-induced cholecystitis is a rare and peculiar entity of vesicular inflammation that is difficult to diagnose and has a high rate of complications and mortality [3, 6].

It occurs in 0.2 to 1% of intensive care patients between the ages of 50 and 70, with a male predominance and a high mortality rate estimated at between 12 and 42% [1, 3]. This prevalence rises to 3.5% in severely burned patients (damage to more than 30% of the body surface) and predominates in young subjects between 30 and 40 years of age [3, 7].

The pathophysiology is poorly understood: the incriminating risk factors are biliary stasis (favored by prolonged fasting, parenteral nutrition, massive transfusions, mechanical ventilation, morphinics, etc.), infection (endotoxin release in septic states), and ischaemia-reperfusion phenomena (due to states of shock, use of catecholamines, severe burns, etc.) [3, 4, 6, 7]. Some authors question the lithiasis origin, which could be the etiology in approximately 50% of stress cholecystitis cases [3].

Clinical symptoms include pain in the right hypochondrium, nausea, and vomiting. Jaundice may be associated, especially in cases of lithiasis leading to obstruction of bile flow. This presentation is often bastardized in multi-tarred patients in the intensive care setting, making diagnosis difficult; confusion with a worsening clinical condition or the appearance of signs of sepsis may be the only findings [3].

Biology reveals a non-specific inflammation syndrome (hyperleukocytosis or leukopenia with elevated CRP) and should look for increased liver enzymes (ASAT, ALAT, gamma-GT, and PAL) or bilirubin, reflecting cholestasis or liver failure in the context of sepsis [3]. These biological disturbances are therefore not specific, especially in the context of extensive burns, given the associated systemic inflammatory response syndrome [7].

Ultrasound is the first-line radiological examination. It shows vesicular distension, laminated parietal thickening, parietal air bubbles, biliary stasis (vesicular sludge), and peri-vesicular effusion [3, 6]. CT scans are mainly used in difficult patient conditions (obesity, meteorism, etc.) to rule out a differential diagnosis or to search for complications (gangrene, perforation). Scannographic signs include hydrocholecyst, dense vesicular content, thickening with parietal enhancement or contrast defect, the presence of parietal air bubbles, and peri-vesicular effusion [3].

Hepatobiliary scintigraphy can be used to diagnose alithiasic cholecystitis by showing the absence of marker fixation (technetium-99m, cholecystokinin, morphine), but it requires patient transport to a nuclear medicine department and a prolonged examination time (from 1 to 6 hours), which limits its use [3, 6].

The presence of an embedded lithiasis in the gallbladder neck or cystic duct is an element of certainty, whereas its absence (on ultrasound or CT scan) makes the diagnosis more difficult, given the frequency of false positives in the intensive care setting. Huffman et al. have proposed radiological criteria for the diagnosis of alithiasic cholecystitis, represented in Figure 3 [6]:

Modality	Criteria		Diagnosis
US	Major	3.5- to 4-mm (or more) thick wall (if at least 5-cm distended longitudinally with no ascites or hypoalbuminemia) Pericholecystic fluid (halo)/subserosal edema Intramural gas Sloughed mucosal membrane	2 major or 1 major and 2 minor (most studies have favored the diagnostic triad—wall thickness, sludge, hydrops)
	Minor	Echogenic bile (sludge) Hydrops = distension greater than 8-cm longitudinally or 5-cm transversely (with clear fluid)	
CT	Major	3- to 4-mm wall thickness Pericholecystic fluid Subserosal edema Intramural gas Sloughed mucosa	2 major or 1 major and 2 minor
	Minor	Hyperdense bile (sludge) Subjective distension (hydrops)	

Figure 3 : Ultrasound and CT diagnostic criteria for acute alithiasic cholecystitis according to Huffman et al.

Half of patients will develop vesicular gangrene, and 10% will progress to perforation; the latter occurs most frequently in subjects with cardiovascular, diabetic, or immunocompromised risk factors, preferentially in the vesicular fundus [2, 3, 5].

Niemeier's 1934 classification of gallbladder perforations into three types [1, 8]:

- **Type 1:** acute perforation in the free peritoneum, often at the level of the vesicular fundus. Clinical findings in this case are similar to those of peritonitis, and diagnosis is based on clinical and radiological data. Treatment is based on surgical exploration with cholecystectomy or cholecystotomy.

- **Type 2:** sub-acute perforation and peri-vesicular abscess formation Diagnosis can be made by ultrasound, CT, or bili-MRI. Treatment depends on the patient's general condition; percutaneous drainage should be performed first in multi-tar patients, with simultaneous cholangiography to look for lithiasis in the main bile duct. Cholecystectomy may be performed immediately if the patient's general condition allows or after percutaneous drainage.

- **Type 3:** chronic perforation and formation of a bilio-digestive fistula. This type predominates in elderly patients with known vesicular lithiasis and may manifest as biliary ileus. The biological work-up is minimally disrupted, and treatment is based on cholecystectomy with excision of the fistulous tract if the patient's condition permits.

In our case, vesicular perforation occurred directly in the peritoneal cavity (Niemeier type 1), complicating alithiasic cholecystitis. Extensive skin burns represented a major stress state and the main risk factor in this young patient.

Induction in a severely burned patient should be undertaken in the same way as for a patient with a full stomach. Anesthesia must take into account the following particularities:

- **Hemodynamic and respiratory particularities:** Hypovolemia is an inevitable consequence of severe burns. If effective filling is delayed, the patient may progress to hypovolemic shock. The latter must be provided either by peripheral venous outlets (16 or 14 gauge at best) or by a central venous line (in the event of peripheral vasoconstriction) to ensure adequate perfusion and compensate for fluid losses. The alteration of plasma proteins and the massive use of crystalloids in burn victims increase the diffusion of plasma into the interstitial sector and the formation of edema. This situation most often calls for the use of colloid or lactated Ringer's filling and sometimes the administration of albumin, which has a high oncotic power [10, 11]. Monitoring of diuresis and invasive blood pressure enables us to assess the effectiveness of filling and detect sudden variations in blood volume (which may be exacerbated by intraoperative hemorrhage due to coagulation disorders) [10]. Internal burns, when associated, often lead to massive airway oedema, particularly of the epiglottis and larynx. In such cases, mask ventilation becomes difficult, and intubation must be performed rapidly, sometimes under fibroscopic control [10]. Emergency tracheostomy remains an alternative if intubation is impossible.

- **Pharmacological characteristics of anesthetic agents:** Burns alter the pharmacokinetics of anesthetic drugs, depending on the extent and depth of skin lesions. Pharmacodynamics are also altered in this setting; there is tolerance to ketamine and thiopental and resistance to the muscle relaxant action of non-depolarizing curares [10]. Ketamine is particularly used in burn patients as it has a sympathomimetic effect (increasing heart rate, blood pressure, and cardiac output). However, its negative inotropic effect necessitates a reduction in the dose administered (0.5 to 1.5 mg/kg) [10]. The use of depolarizing curares is contraindicated beyond 48 to 72 hours after burns, as there is a significant risk of hyperkalemia and rhythm disorders [12].

IV. Conclusion:

Vesicular perforation in alithiasic stress-induced cholecystitis remains a rare, serious, and potentially life-threatening complication with an unspecific clinical and biological presentation. Imaging can contribute to the diagnosis, and the therapeutic attitude depends on the type of perforation and the patient's condition. Anaesthesia in severely burned patients must take consideration of the particular features of this situation.

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