Acute Cholecystitis as Cause of Death after Surgery for Lumbar Canal Stenosis

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Post-surgical acute cholecystitis has been described mainly as a complication of major abdominal or thoracic surgery sometimes associated with musculoskeletal surgery. In spine surgery it has been related to large-scale procedures such as the correction of deformities in adults.
The most frequently mentioned risk factors are hydric restriction, fever, hemolytic phenomena, multiple blood transfusions, nutritional disorders, certain drugs (anesthetics, codeine. atropine, meperidine, morphine) and hemodynamic alterations. The risk is especially high when several of the factors above co-occur in a single patient subjected to a physically stressful situation like surgery.
Diagnosis of acute postoperative cholecystitis is often challenging since the condition is often marked by the initial surgical procedure. Symptoms like fever, leucocytosis and abdominal pain after a favorable immediate post-op should put us on guard.
Sonographic confirmation and the quick implementation of appropriate treatment are the only way of reducing the high death toll of this complication.

Key words: *cholecystitis, lumbar spine, early diagnosis, mortality.*

Colecistitis aguda como causa de muerte tras cirugía por estenosis del canal lumbar

La colecistitis aguda posquirúrgica se ha descrito fundamentalmente como una complicación de cirugía mayor abdominal o torácica, y en ocasiones asociada a cirugía del aparato locomotor. En cirugía del raquis se ha asociado a grandes procedimientos como la corrección de deformidades en el adulto. Los factores de riesgo más reconocidos son la restricción hídrica, fiebre, fenómenos hemolíticos, trasfusiones sanguíneas múltiples, trastornos nutricionales, fármacos (anestésicos, codeína, atropina, meperidina, morfina) y alteraciones hemodinámicas. Existe un riesgo especialmente elevado cuando confluyen varios de ellos en un paciente sometido a una situación de estrés físico como la cirugía.
El diagnóstico de la colecistitis aguda posoperatoria suele ser difícil, al quedar frecuentemente enmascarado por el procedimiento quirúrgico inicial. Deberá existir un alto índice de sospecha ante todo paciente que tras un posoperatorio inmediato favorable desarrolle fiebre, leucocitosis y dolor abdominal.
La confirmación ecográfica y la instauración rápida del tratamiento oportuno son el único modo de disminuir la elevada tasa de mortalidad de esta complicación.

Palabras clave: *colecistitis, raquis lumbar, diagnóstico precoz, mortalidad.*

Population ageing, the sophistication of diagnostic methods and the optimization of surgico-anesthetic techniques have led to the sustained increase in the prevalence of lumbar arthrodesis in industrialized societies[1].

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Acute cholecystitis is a postsurgical complication with a devastating presentation and a high death toll, usually associated with major abdominal and thoracic surgery as well as major orthopedic procedures[2-8]. In spine surgery it has been described as an unusual complication in the correction of deformities[9,10]. Up to the present day, we have found only one case of acute cholecystitis after lumbar laminectomy in the literature.

We here present the case of a patient who was operated for lumbar canal stenosis by decompression and instrumented posterolateral arthrodesis, and died on the third week after surgery as a consequence of acute postsurgical cholecystitis.

CASE REPORT

We analyze the case of a male patient of 72 years of age, in good general health and with a history of asymptomatic duodenal ulcus, arterial hypertension and decompressive cervical surgery for myelopathy three years before.

He consulted us due to pain in the two lower extremities, predominantly on the right, and because standing and walking produced neurogenic claudication and a walking perimeter of under 200 meters. Physical exploration showed a right S1 radiculopathy with hypostasis in the soles of the feet, abolition of the achilleous reflex and paresis (3/5) of the right sural triceps. Complementary exploration showed lumbar canal stenosis in segments L3-L4 and L4-L5 (figs 1-5). The electromyographic study found S1 bilateral denervation, predominantly on the right side.

The patient was evaluated preoperatively and operated, with general anesthesia and in the prone position. Decompression of segments L3-L4 and L4-L5 was carried out by means of bilateral L3 and L4 laminotomy associated to instrumented posterolateral L3-L5 arthrodesis, and using pedicular instruments and a cortico-cancellous autologous-homologous graft combination. The surgery lasted 180 minutes and preoperative bleeding was 800cc. No complications or alterations of vital constants were encountered during the procedure (fig 6).

Twenty-four hours after surgery, the patient was sent to a hospital bed. He was hemodynamically stable, without neurologic deficit and pain satisfactorily under control. Forty-eight hours later, his body temperature was normal, he was stable, and the surgical wound in a satisfactory state. Consequently, suction drainage was removed and both sitting and standing were allowed.

On the third day post-op, the patient presented with epigastric pain accompanied by sweating and hypotension. On being examined by the internal medicine service, he was diagnosed with myocardial ischemia and sent to the Intensive Care Unit (ICU) on a diagnosis of cardiogenic shock, acute coronary syndrome and cardiac arrhythmia due to atrial fibrillation. The patient responded favorably

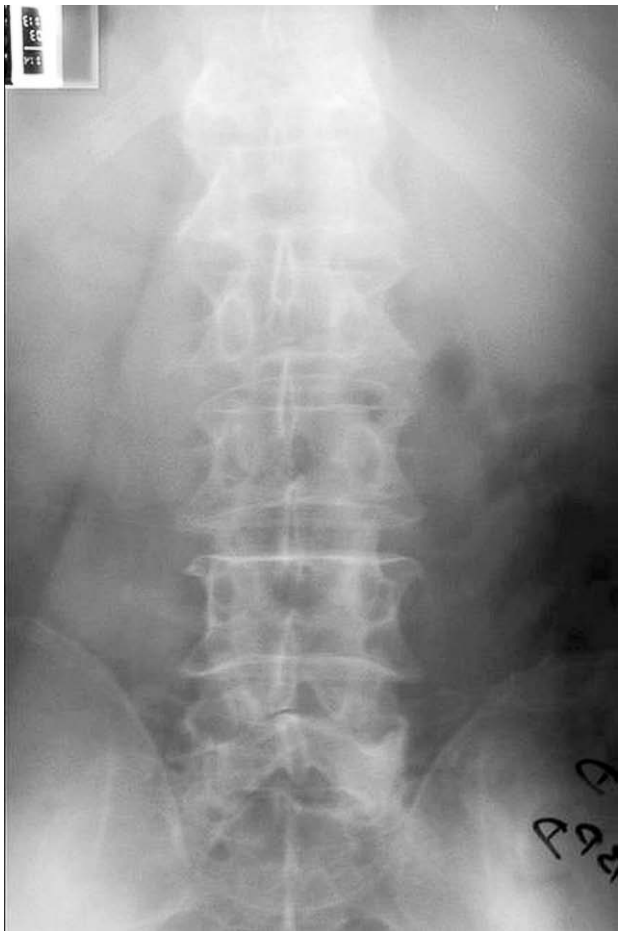

Figure 1. *Preoperative posteroanterior x-ray*

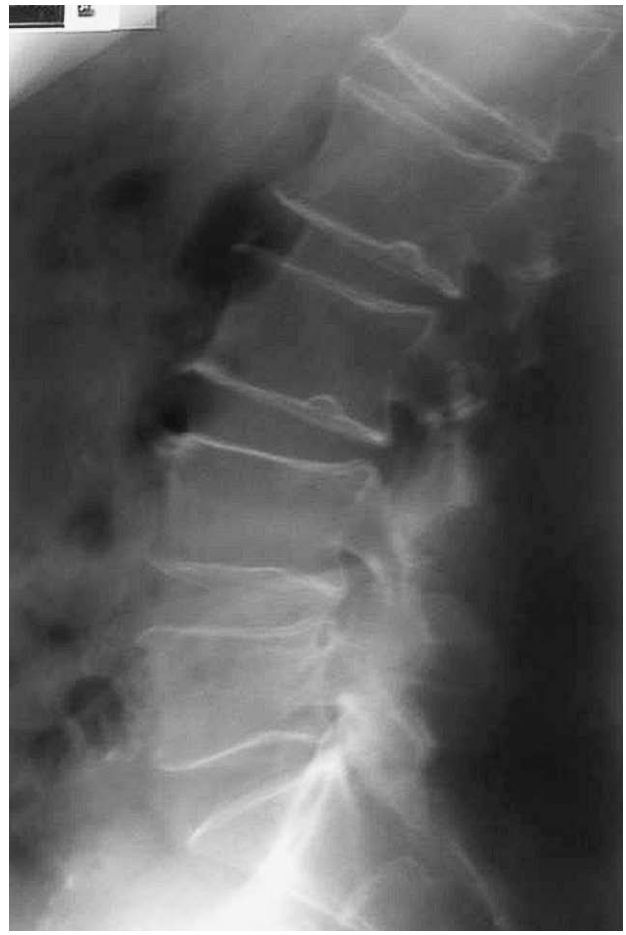

Figure 2. *Preoperative lateral x-ray.*

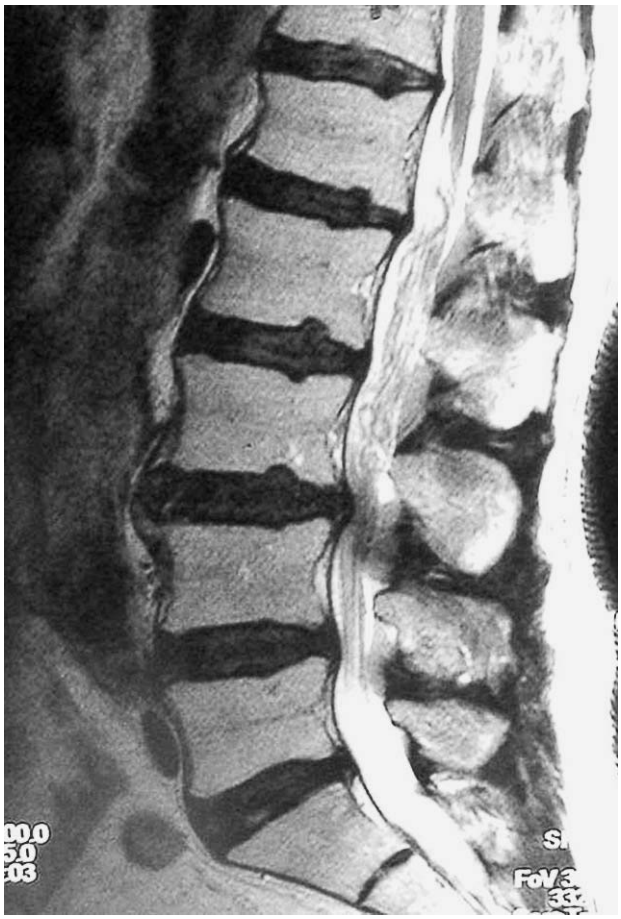

Figure 3. *Sagittal MRi showing spinal canal stenosis mainly at levels L3-L4.*

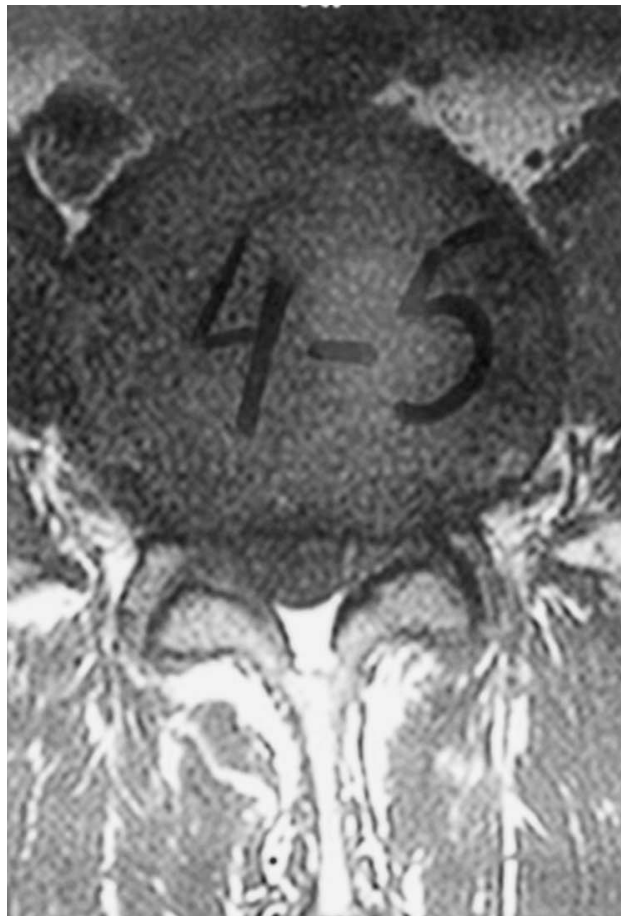

Figure 5. *Axial MRi showing canal stenosis at levels L4-L5.*

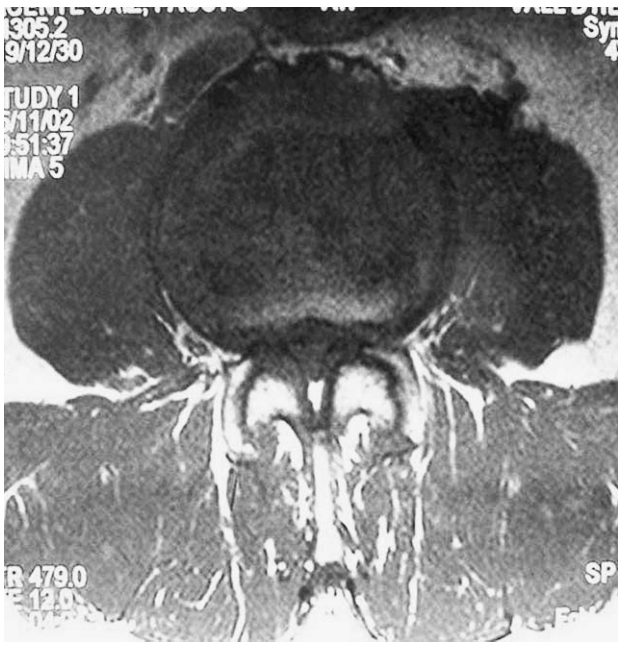

Figure 4. *Axial MRi at level L3-L4 showing intraforaminal stenosis.*

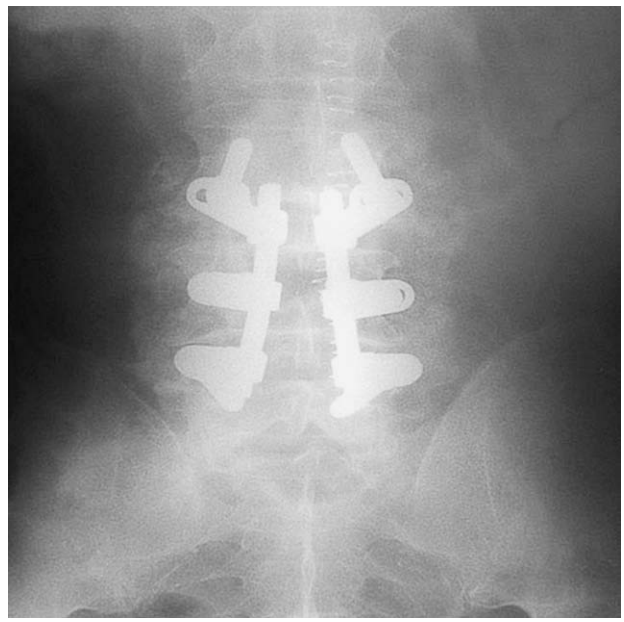

Figure 6. *Preoperative posteroanterior x-ray showing bilateral L3 and L4 laminotomy associated to instrumented posterolateral L3-L5 arthrodesis.*

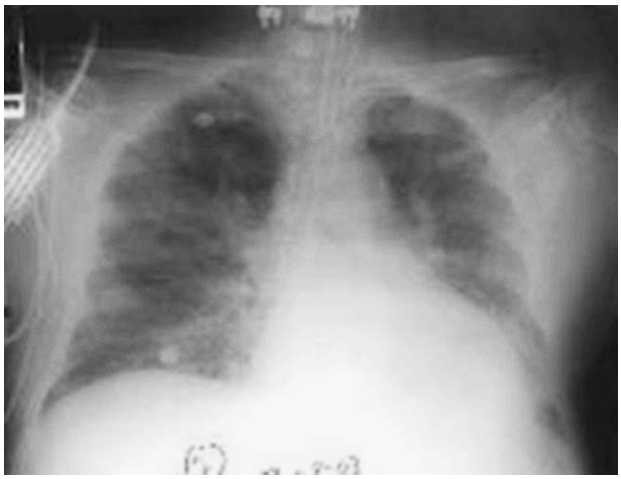

Figure 7. *Anteroposterior thorax x-ray showing bilateral lung infiltration.*

to the assigned medical treatment and was transferred to a hospital bed 24 hours after his entry into the ICU with a normal electrocardiogram (ECG). On the fifth day after surgery, the epigastric pain reappeared although this time it was transfixing and continuous and irradiated to the right hypochondrium. Physical exploration found hepatomegalia with a positive Murphy's sign. Laboratory post-op data was normal (3.4 10 x 6 red corpuscles; 10.7 hemoglobin; 31.4% hematocrits; 8,700 white corpuscles and 198,000 platelets). Abdominal ultrasound showed an edematous gallbladder, with a 3.5mm-thick wall, lithiasis at the level of the gallbladder neck and the presence of perivesicular liquid. The ultrasound Murphy's sign was positive (the abrupt pressure release caused by the transducer at the level of the gallbladder triggers off pain). In consequence, the patient was diagnosed with acute postsurgical cholecystitis.

Once the patient had been evaluated by the general surgery service, a stand-by was decided on due to the fact that there had recently been an episode of ischemic cardiopathy. Antibiotic treatment with metronidazol (500 mg/8 hours) and gentamin (80 mg/8 hours) was administered. On the seventh day after surgery the lumbar wound showed signs of dehiscence, due to which the patient was operated again for debridement and cleansing. In the deep cultures of the lumbar wound *E. Coli* was found. The patient could not be extubated because he presented with a breathing deficiency associated to a bilateral lung infiltration. The bronchial suction was also positive for *E. Coli* (fig. 7). The general condition of the patient deteriorated and the abdominal distension persisted. Because of this a cholecystectomy was performed.

The laparotomy showed the presence of a septic plastron with intra-abdominal liquid contaminated with *E. Coli* and multiple biliar lithiasis. Postoperatively the patient developed septic shock due to *E. Coli* and respiratory distress. He died three weeks after the surgery for lumbar canal stenosis.

DISCUSSION

Acute postsurgical cholecystitis presents a rate of occurrence that ranges between 22 and 63%, considering all surgical specialities[10]. It may occur in 0.9% of the total number of multi-trauma patients[11], and in 1.6% of the total number of patients in a critical state[12]. In orthopedic surgery it has been mainly associated with hip arthroplasty, open reduction and internal fracture fixation, hemipelvectomy and spinal fusion for the correction of deformities at any age[10]. A wide range of prevalence rates are mentioned in the literature, which go from 16.5 to 37%[3,13]. It is more frequent in males (7:1) and in its alithiasic form. It usually has a late diagnosis and therefore treatment is initiated when the disease is at an advanced stage and with necrotic changes, gangrene and perforation. In such advanced phases it bears a mortality rate of 75%[4,6-8].

The etiopathogeny of acute postsurgical cholecystitis has not yet been satisfactorily determined. However, it is agreed that there exist a series of risk factors that can trigger it when they are associated with situations in which the patient undergoes a physically stressful situation, such as surgery[11]. Fever, dehydration, hydric restriction[2], multiple transfusions and hemolytic phenomena[5] can increase gall concentrations. Other factors that can increase gall concentrations are prolonged fasting and parenteral feeding due to a lack of stimulus for carrying out the release of cholecystoquinine[7,12,15]. The increase of gall concentrations would seem to favor the appearance of edema with venous and lymphatic occlusion of the bladder wall, which would help cause a severe inflammatory reaction which in turn assists the progress of necrosis and perforation[7]. The use of drugs, anesthetics and prolonged mechanical ventilation may hinder the emptying of the bladder[12,15-19]. Likewise, medication such as atropine, codeine, meperidine or morphine occlude the gall tract, stimulating the contraction of the Oddi sphincter[4,11]. Liver and bladder hypoperfusion due to hypotension or hemodynamic shock can cause focal necrosis of the bladder mucosa[6].

Approximately 50% of the total number of postsurgical cholecystitis will develop with overinfection of the gall tract[4]. There are different hypotheses attempting an explanation of the bacterial colonization of this necrotized gall tract.

When cholecystitis appears in a patient with a known septic focus (war wounds, compound fractures), the contamination of the gall tract originating in this infectious focus seems to be the most probable outcome[11,20]. On the other

hand, the absence of a recognizable septic focus in many cases[4] and the protagonism of the gram-negative bacilli of digestive origin suggest a colonization of the gallbladder, necrotized by the habitual digestive flora.

Early diagnosis and treatment are the most important factors to take into account with a view to reducing the high death toll of this complication[4,6,7], which tends to appear within the first week and month after surgery[8,22]. It is often related to oral reintroduction following prolonged fasting, when a sudden liberation of cholecystoquinine is produced, causing energetic bladder contraction[4,7,9,23]. In many cases the symptoms indicating the presence of this complication are non-specific and do not reflect the severity of the disease[3,11]. There is often pain in the top right abdominal quadrant, with sudden onset (25%), and with rigidity and signs of peritoneal irritation. In up to 42% of the cases it may appear with a palpable mass at this level[13,4,11].

The symptom that appears initially with the highest degree of frequency is fever. If it is persistent it must be considered a highly suspicious sign[11,12]. Laboratory findings comprise leukocytosis, an increase of bilirubin, and alkaline phosphates in the bloodstream[3,4,22]. Liver tests are usually normal[11]. Simple X-ray images may show pleural effusion, left diaphragm elevation and dilatation of the small intestine due to paralyzing colic[3,4]. Intravenous cholangiography is not recommended as a diagnostic method due to the toxicity of the contrast material and its deleterious effect on the liver[23].

Ultrasound is deemed to be the best diagnostic method[3]. It has shown an increase in the size of the bladder and a thickening of its wall[11]. Axial CT-scanning may reveal the existence of bladder distension with edema and perivesicular abscess[11]. Although not initially recommended, it may prove helpful in the presence of intestinal gas[21].

In septic patients, treatment may begin with intensive fluid therapy associated with antibiotic therapy to prevent bacterial colonization of the bladder[10,17]. Considering the high incidence of gangrene and perforation, we suggest cholecystectomy as the best treatment. This kind of surgery has a high degree of tolerance and postoperatively usually develops without complications[4].

Offloading cholecystostomy as an alternative treatment seems to be unsatisfactory because of the presence of inflammatory phenomena and a high level of gall viscosity[8]. It could be recommendable as a palliative method to be used in patients in a critical state[12]. Laparotomy findings show an edematous and distended bladder with fibrous adhesions to adjacent organs. Bladder content reveals an increase in viscosity and a dark green colour[9]. The presence of gallstones is exceptional[14,7,12,17,24,25].

No practical method exists that will prevent the appearance of postsurgical cholecystitis. Therefore, an exhaustive control of risk factors is exceedingly necessary–particularly in the case of elderly patients–as well as an early surgical determination on encountering signs of the appearance of this complication[7,8].

In conclusion, population ageing is related to an increase in the practice of decompressive lumbar surgery. Acute postsurgical cholecystitis is a potentially deadly complication, which may appear in the postsurgical stage of decompressive lumbar surgery in adults. The existence of risk factors related to acute postsurgical cholecystitis demands exhaustive control of elderly patients. Early diagnosis (ultrasound), in the face of the clinical deterioration of a patient with normal immediate postsurgical evolution is crucial to reach a satisfactory resolution of this complication.

REFERENCES

1. Deyo RA, Nachemson A, Mirza SK. Spinal fusion surgery. The case for restraint. N Engl J Med. 2004;350:722-6.
2. Thompson JW et al. Acute cholecystitis complicating operation for other diseases. Ann Surg. 1962;155:489.
3. Abrahamson J, et al. Acute cholecystitis after orthopaedic operations. Inter Orthop. 1988;12:93-5.
4. Lindberg EF, et al. Acalculous cholecystitis in vietnam casualties. Ann Surg. 1970;171:152-7.
5. Robertson RD. Noncalculous acute cholecystitis following surgery trauma and illness. Am Surg. 1970;36:610.
6. Winegagner FG, et al. Postraumatic acalculous cholecystitis. A highly lethal complication. J Trauma. 1971;11:567.
7. Ottinger LW. Acute cholecystitis as a postoperative complication. Ann Surg. 1976;184:162-5.
8. Du Priest RW, et al. Acute cholecystitis complicating trauma. Ann Surg. 1979;189:84-9.
9. Floman Y, et al. Acute cholecystitis following the surgical treatment of spinal deformities in the adult. Clin Orthop. 1980;(151):205-9.
10. Susan H, Westfall MD, et al. Acute cholecystitis after spinal fusion and instrumentation in children. J Ped Orthop. 1991; 11:663-5.
11. Heruti R, et al. Acute acalculous cholecystitis as a complication of spinal cord injury. Arch Phys Med Rehabil. 1994; 75:822-4.
12. Long TN, et al. Acalculous cholecystitis in critically ill patients. Am J Surg. 1978;136:31-6.
13. Devine RM, et al. Acute cholecystitis as a complication in surgical patients. Arch Surg. 1984;119:1389-93.
14. Knudson RJ, et al. Acute cholecystitis in the postoperative period. New Eng J Med. 1963;269:289.
15. Glenn F, et al. Acute cholecystitis following the surgical treatment of unrelated disease. Surg Gynecol Obstet. 1956;102: 145-53.
16. Detrie PH. Acute cholecystitis after an operation without relation to the biliary tract. Presse Med. 1957;65:527.
17. Jönsson PE, et al. Postoperative acute acalculous cholecystitis. Arch Surg. 1976;111:1097-101.
18. Johnsson EE, et al. Continuous positive pressure ventilation and choledochoduodenal flow resistance. J Appl Physiol. 1975;39:937.
19. Ullman M, et al. Postraumatic and postoperative acute acalculous cholecystitis. Acta Chir Scand. 1984;150.

20. Weeder RS, et al. Acute noncalculous cholecystitis associated with severe injury. Am J Surg. 1970;119:729-32.
21. Herlim P, et al. Acute acalculous cholecystitis following trauma. Br J Surg. 1982;69:475-6.
22. Howard RJ. Acute acalculous cholecystitis. Am J Surg. 1981; 141:194-8.
23. Johnson HC, et al. Intravenous cholangiography of the gallbladder in the differential diagnosis of acute cholecystitis. Radiology. 1960;74:790-6.
24. Munster AM, et al. Acalculous cholecystitis in burned patients. Am J Surg. 1971;122:591-3.
25. Brenner RW, et al. Cholecystitis in children. Rev Surg. 1964; 21:327-35.

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