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A MISUNDERSTOOD INTESTINAL PERFORATION BELIEVED ACUTE PANCREATITIS: A CASE REPORT

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ABSTRACT

Acute pancreatitis represents one, possible but rare, of the several complications of laparoscopic cholecystectomy. In the case reported, a 31-year-old female patient complained of abdominal pain after laparoscopic cholecystectomy. The clinical picture, the high values of serum amylase, lipase and white blood cell count and the subsequent abdominal computed tomography (CT) led to diagnose an acute biliary pancreatitis. This was pharmacologically treated, but the patient worsened in a few days. A contrast-enhanced CT showed the presence of free air and effusion into the peritoneal cavity. The patient was submitted to another intervention, which revealed a 1-cm jejunal perforation. The injured loop was then repaired and the patient discharged after three days. The cause remains obscure but it was likely due to umbilical trocar insertion. An upper quadrant abdominal pain with elevated amylase and lipase serum concentration, not always indicate the presence of an acute pancreatitis but could be associated to a difficult case of intestinal perforation.

***Key words**: Cholecystectomy, intestinal perforation, pancreatitis.*

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Introduction

Acute pancreatitis is an acute inflammatory process with multiple aetiologies. In Italy, the incidence ranges from 20 to 40 cases out of 100,000 inhabitants/year and the mortality rate ranges from 5 to 10%[1]. In the most of series published in medical literature, gallstones are the leading cause, followed by alcohol. The frequencies of metabolic and infectious causes in the present series are higher than those reported[2-4]. Less common causes are represented by drugs, toxic-metabolic disorders and iatrogenic causes (Tab.1). Laparoscopic cholecystectomy can be included among the iatrogenic causes of pancreatitis, especially when the patient is affected by gallbladder microlithiasis. In fact, in this case, manoeuvres performed for cholecystectomy can potentially mobilize gallstones, allowing them to obstruct the main biliary duct. Pancreatitis often presents with a characteristically dull, boring, and steady abdominal pain, usually sudden in onset and gradually becoming more severe until reaching a constant ache; it is most often located in the upper abdomen and may radiate directly through to the back. Nausea, vomiting, sometimes with anorexia, and diarrhoea are also present. Serum amylase and lipase levels are typically elevated in persons with acute pancreatitis. In research studies, amylase or lipase levels at least 3 times above the reference range are generally considered diagnostic of acute pancreatitis. Serum amylase determinations are routinely available, but they are not specific for pancreatitis. Elevations can occur in patients with small intestinal obstruction, mesenteric ischemia, tubo-

ovarian disease, renal insufficiency, or macroamylasemia. Rarely, elevations may reflect parotitis. The serum half-life of amylase is short, and elevations generally return to the reference ranges within a few days. Lipase has a slightly longer half-life and its abnormalities may support the diagnosis if a delay occurs between the pain episode and the time the patient seeks medical attention. Elevated lipase levels are more specific to the pancreas than elevated amylase levels. Lipase levels remain high for 12 days. In patients with chronic pancreatitis (usually caused by alcohol abuse), lipase levels may be elevated in the presence of a normal serum amylase level. We report a case of acute abdominal wall in a young adult female patient who underwent laparoscopic cholecystectomy, one day after intervention. Due to the clinical picture and the high levels of amylase and lipase, it was considered a case of acute biliary pancreatitis, and therefore treated with conservative medical therapy. Unfortunately, it was a case of bowel perforation of unknown origin.

Aetiology of acute pancreatitis	%
Gallstones	40-70 %
Alcoholism	15-26 %
Surgery	9-12 %
Idiopathic	5-7 %
Post-traumatic	1,5-5 %
Other:	5-8 %
• CPRE	
• Ductal obstruction	
• Hypercalcemia	
• Drugs	
• Infections	
• Vascular factors	
• Pregnancy	

Table 1: Possible causes of acute pancreatitis.

Case presentation

A 31-year-old woman was admitted to our hospital with right upper abdominal pain, also referred at the right scapula. Her body temperature was normal, but she complained of nausea and referred she vomited the night before. The patient reported the onset of the symptoms two years before, during her pregnancy, and laboratory tests revealed an increase of her transaminases (AST: 51 U/L, ALT: 68 U/L). Patient's abdomen was not distended, soft and non-tender; deep palpation evoked an intense pain in the right upper quadrant; Murphy's sign was positive, Blumberg's negative. Bowel sounds were present. The patient was healthy and did not refer anything particularly relevant in her medical history. Interestingly, her mother underwent cholecystectomy about ten years before. Laboratory test showed no abnormalities (Table 2), but abdominal ultrasonography revealed multiple stones in gallbladder with no dilation of the biliary tree. The patient was submitted to laparoscopic cholecystectomy. The gallbladder appeared with thickened walls, swollen and bent. Dissection manoeuvres to detach the gallbladder from the liver, were laborious due to the presence of many and strong adhesions between the two organs. At the end of the operation, before trocar extraction procedure, the abdominal cavity was carefully examined and any fluid collection aspirated. In the afternoon, the patient complained of intense, crampy and constant abdominal pain (Visual Analogue Scale - VAS: 8/10), treated with intramuscular administration of ketorolac trometamina 30 mg/ml and butyl-scopolamine 20 mg/ml.

The patient did not show any clinical improvement after the treatment. A 30 ml of biliary fluid from the drainage tube was observed. The patient presented an intense abdominal pain, more annoying in the upper quadrants, radiating to the back; the abdomen was distended, poorly treatable, hypoperistaltic, painful on deep palpation. Blumberg's sign was positive. Abdominal computed tomography (CT)-scan without contrast reported the presence of free effusion at the gallbladder lodge, at the perihepatic and perisplenic areas, and in the pelvic cavity, and the absence of other significant lesions to other abdominal organs (Fig. 1). The first control of blood tests showed an increase in amylase (259 U/L - Normal values: 28-100 U/L), whereas white blood cells (WBC) were normal (Tab. 2). Blood exams carried out in the following four days, clearly showed a progressive and significant increase of amylase and lipase (Fig. 2), an increase of WBC with neutrophilia and lymphocytosis (Fig. 3), and a progressive increase of transaminases too. The increase of the pancreatic enzymes (Tab. 2), symptoms and abdomen CT, led to a diagnosis of acute pancreatitis, which was pharmacologically treated with 2 ml octreotide three times daily in combination with intravenous levofloxacin 500 mg/day and betamethasone 4 mg/2 ml daily. Being all data suggestive for an acute pancreatitis, the patient was

	Pre-operative values	Post-operative days								
		1st	*2nd*	*3rd*	*4th*	*5th*	*6th*	*7th*	*8th*	*9th*
BILIRUBIN (mg/dl)	0.35	1.22	0.98	1.17	1.73	1.59	0.99	1.19	1.04	0.79
Conjugated	0.09	0.5	0.61	0.61	1.14	0.97	0.6	0.59	0.39	0.5
Unconjugated	0.26	0.72	0.37	0.56	0.59	0.62	0.39	0.6	0.65	0.29
α-AMYLASE(U/L)		569	722	372	818	277	216	183	138	156
LIPASE(U/L)		37	30	67	106	158	322	318	301	287
γ-GT(U/L)	28	33	23	22	34	54	45	47	48	33
WBC (x103/µl)	4.83	4.98	7.35	8.76	9.87	12.82	9.19	8.82	7.64	10.6
NEUT (x103/µl)	2.99	4.98	6.6	7.25	9.02	11.7	7.1	6.94	5.65	8.58
LYM (x103/µl)	1.48	0.33	0.43	0.52	0.6	0.57	1.36	1.22	1.33	1.29
AST/ALT(U/L)	21/28	36/56	25/38	18/43	18/23	17/41	17/42	15/38	13/33	25/27

Table 2: Patient's blood exams from first to ninth postoperative day.

placed on an empty stomach with daily control of blood pressure, cardiac frequency and temperature. Three days after intervention, an abdominal contrast-enhanced CT was performed, due to the recrudescence of the abdominal pain (VAS: 9/10).

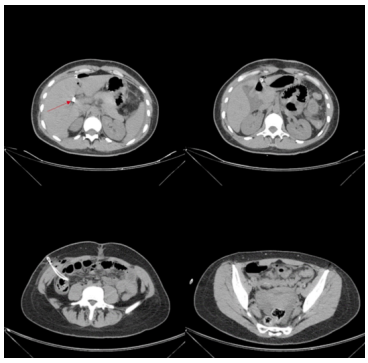

Figure 1: Abdomen CT. Presence of free fluid into the gallbladder lodge, involving the perihepatic and perisplenic areas. Mild pneumoperitoneum. Red arrow indicates the metallic clips used in laparoscopic cholecystectomy.

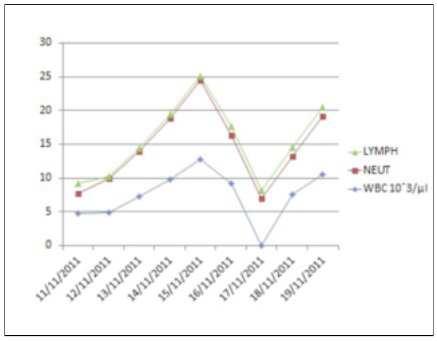

Figure 2: White Blood Cell, lymphocyte and neutrophil trend during hospitalization.

The pain was refractory to pain killers (Paracetamol 1000 mg and Ketorolac Tromethamine 30 mg/ml). Patient's abdomen was bloated and tender. Blumberg's sign was highly positive. The abdominal drainage was strangely empty. CT showed an increase of the free air in the peritoneal cavity, ascites distributed in the perihepatic zone, in the perihepatocolic recesses and in the pelvic cavity, and a gaseous distension of the small intestine loops with air-fluid levels. Pancreas had a normal aspect with no dilatation of Wirsung duct (Fig. 4).

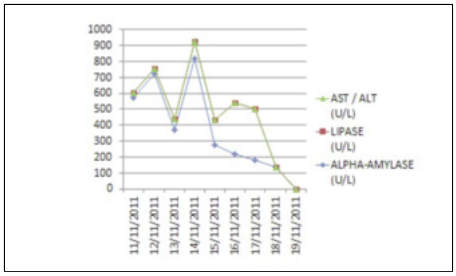

Figure 3: Pancreatic and hepatic enzymes during hospitalization.

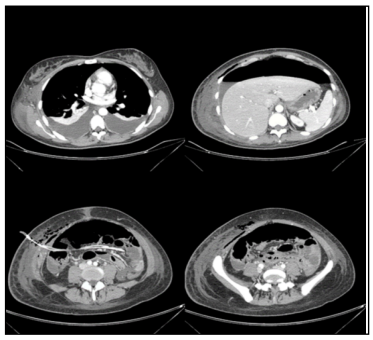

Figure 4: Contrast-enhanced abdominal CT, three days after cholecystectomy. Increase of free air in the peritoneal cavity; ascites distributed in the perihepatic zone, in the perihepatocolic recesses and in the pelvic cavity; gaseous distension of the small intestine loops with air-fluid levels. Pancreas of normal aspect with no dilatation of Wirsung duct.

Symptoms were therefore attributable to an intestinal perforation with chemical peritonitis. For this reason, four days after cholecystectomy, the patient underwent an explorative laparotomy, which revealed a perforation of a jejunal loop, 60 cm far from Treitz ligament. The greenish abdominal liquid was drained, the abdominal cavity washed with warm saline and the cleft closed with mucosal non-absorbable 2/0 suture, reinforced with sero-serous non-absorbable 2/0 stitches. In the following post-operative days, there was a marked improvement in laboratory values (Tab. 2), with a return to normal range of white blood cells and pancreatic enzymes, and a general improvement of patient clinical conditions. The patient was discharged after five days (nine days after cholecystectomy), in very good clinical conditions..

Discussion and conclusion

The presented case one more time shows how difficult is to make a diagnosis in medicine. Often, many confounding factors can mask the classical presentation of a pathologic condition, and lead to a wrong final decision. In the case reported many elements led us to hypothesize the occurrence of a post-operative pancreatitis. The clinical picture, amylase, but, overall, lipase serum levels, and abdominal CT seemed to speak out: an upper quadrant abdominal pain radiating to the back, amylase or lipase levels at least 3 times above the reference range, and radiologic images compatible with a picture of postoperative laparoscopic cholecystectomy, could be considered enough to make a diagnosis of pancreatitis. Moreover, the small amount of yellowish fluid into the drainage bag is not so helpful to hypothesize something different, especially whether it results empty, as it presented in the days following operation. Contrast-enhanced CT-scan, performed three days after cholecystectomy, depicted a clear picture of intestinal perforation, with free air and fluid in peritoneal cavity. The subsequent explorative laparotomy revealed a one cm perforation of a jejunal loop, 60-cm-far from Treitz ligament.

The occurrence of a delayed intestinal perforation is well described in literature[5]. Laparoscopic cholecystectomy can be associated with bowel injury, almost always caused by injudicious and careless use of electro-cautery. Since these injuries are less common, they are not thought of and because they are usually thermal, they present late and are often missed. Usually, duodenum or colon are the most favourite targets of electrothermal injuries[6]. A jejunal perforation due to thermal injury has never been described, and it is difficult to imagine how it would have been occurred. In fact, during cholecystectomy, all manoeuvres are limited to the small area around the organ, and accidental lesions normally occur because of adhesions between the gallbladder and the neighbouring organs (particularly duodenum and colon). A one cm jejunal perforation, 60-cm-far from Treitz ligament, could be only explained by a trocar injury, likely occurred during the initial entry. The bowel perforation was probably present since the first operation, but, unfortunately, it remained unrecognized due to abdomen CT, that was not so clarifier, and elevated serum concentration of amylase and lipase. The latter event, which led us astray, could be explained by peritoneal absorption of the two enzymes, as postulated for bilirubin by Stathakos et al.[7] in the far 1970. Strangely, abdominal cavity exploration, before trocar extraction, did not show any abnormalities of bowels, and did not reveal the presence of blood or stools. Intraoperative Cholangiography (IOC) has an important role in preventing all complications related to biliary tree laparoscopy, depicting perfectly the anatomy of the biliary tree and diagnosing lithiasis of the common bile duct during intervention[8]. Probably, the use of IOC would have helped us to exclude pancreatitis immediately, and focus our attention on bowel perforation. Unfortunately, even if IOC is performed only selectively, many cholangiograms would have to be obtained to find a small number of stones; thus, IOC is not cost-effective for this purpose[9,10].

We reported a case apparently easy to solve but with some diagnostic difficulties. In fact, the clinical and laboratory data were strongly suggestive of acute pancreatitis, a rare but possible complication of video-laparoscopic cholecystectomy. The first CT-scan performed without contrast was a confounding factor leading to a wrong diagnosis of acute pancreatitis. The clinical data and laboratory findings were attributable to the spreading of enteric material through an intestinal perforation, resulting in chemical peritonitis and trans-peritoneal reabsorption of the same enzymes into blood. Any patient, who is not comfortable (sitting up in the bed having her morning cup of tea and wanting to go home), does not have stable vital signs (pulse, temperature and respiratory rate) and does not have a settled (soft and non-tender) abdomen the day after laparoscopic cholecystectomy, should be strongly

suspected to have sustained an intra-operative injury (usually to the bile duct but may be to the duodenum or the colon) and appropriately investigated and managed.

References

1) De Nuzzo D, Miconi A, Pierantognetti P. *Management of suspected acute inflammatory pancreatopathies in a "real-world" setting: a single-centre observational study*. JOP 2014; 15: *175-181*.
2) Birgisson H, Möller PH, Birgisson S, Thoroddsen A, Asgeirsson KS, Sigurjónsson SV, Magnússon J. *Acute pancreatitis:a prospective study of its incidence, aetiology, severity, and mortality in Iceland*. Eur J Surg 2002; 168: *278-282*.
3) de Beaux AC, Palmer KR,Carter DC. *Factors influencing morbidity and mortality in acute pancreatitis; an analysis of 279 cases*. Gut 1995; 37: *121-126*.
4) Kaya E, Dervisoglu A. Eroglu C, Polat C, Sunbul M, Ozkan K. *Acute pancreatitis caused by leptospirosis: report af two cases*. World J Gastroenterol 2005; 11: *4447-4449*.
5) Polychronidis A, Tsaroucha AK, Karayiannakis AJ, Perente S, Efstathiou E, Simopoulos C. *Delayed perforation of the large bowel due to thermal injury during laparoscopic cholecystectomy*. J Int Med Res. 2005 May-Jun; 33(3): *360-3*.
6) Polychronidis A, Tsaroucha AK, Karayiannakis AJ, Perente S, Efstathiou E, Simopoulos C. *Delayed perforation of the large bowel due to thermal injury during laparoscopic cholecystectomy*. J Int Med Res. 2005 May-Jun; 33(3): *360-3*.
7) Stathakos P, Fanska R, Carbone JV, Grodsky GM. *Jaundice associated with peritoneal absorption of bilirubin*. Gastroenterology 1970; 59: *769-778*.
8) Tomasello G, Palumbo VD, Damiani P, Damiani F, Damiano G, Spinelli G, Accardo FM, Lo Monte AI. *Intraoperative cholangiography during laparoscopic cholecystectomy: what role?* Acta Med Mediterr 2012; 28: *281-285*.
9) Nugent N, Doyle M, Mealy K. *Low incidence of retained common bile duct stones using a selective policy of biliary imaging*. Surgeon 2005; 3: *352-356*.
10) Barkun JS, Fried GM, Barkun AN, Sigman HH, Hinchey EJ, Garzon J. *Cholecystectomy without operative cholangiography. Implications for common bile duct injury and retained common bile duct stones*. Ann Surg 1993; 218: *371-379*.

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