CASE REPORT

Laparoscopy-induced severe renal failure after appendectomy

Ignacio Aguirre-Allende1,*, Lander Gallego-Otaegui2, Jose Luis Elosegui-Aguirrezabala1, Carlos Placer-Galán1, and Jose Maria Enriquez-Navascués1

1Colorectal Surgery Department, University Hospital Donostia, San Sebastian, Spain and 2Endocrine Surgery Department, University Hospital Donostia, San Sebastian, Spain

*Correspondence address. University Hospital Donostia, Begiristain Doktorea Pasealekua, 109, 20014 San Sebastian, Spain. Tel: +34 943 00 70 00; Fax: +943 00 72 33; E-mail: ignacio.aguirreallende@osasidetza.eus

Abstract

Laparoscopy has gained importance in the abdominal emergency surgery field. Acute appendicitis is one of the major indications for emergency surgery, being laparoscopy the gold standard approach. We report a case of a 39-years-old female presenting with acute kidney injury (AKI) after laparoscopy. Differential diagnosis was considered with prerenal AKI etiology due to sepsis and low fluid input, however this was ruled out due to absence of electrolyte imbalance and no correlation with septic parameters. Laparoscopy CO2 pneumoperitoneum can potentially lead to multiple organ failure, including renal. Laparoscopy induced AKI is related with both hormonal stimuli for renal vasoconstriction and increased intra-abdominal pressure, causing hypoxemia and tubular renal injury. In conclusion, very few cases of laparoscopy induced AKI in young patients with no previous renal disease have been reported. Surgeons must consider this complication in the differential diagnosis of postoperative AKI.

INTRODUCTION

Laparoscopic approach (LA) has gained importance in abdominal emergency surgery field. Acute appendicitis (AA) is one of the leading causes of acute abdomen and represents one of the most reported interventions in emergency surgery. According to the published literature, laparoscopy is considered the gold standard approach for AA [1]. Laparoscopy implies the creation of a pneumoperitoneum by insufflation of CO2 in the abdominal cavity. Furthermore, this increased abdominal pressure (IAP) leads to both modifications in organ perfusion and stimulation of hormonal systems. All these can potentially produce a multiple organ functional impairment, being kidney dysfunction one of the most representatives. This clinical report highlights potential laparoscopy-induced renal function (RF) impairment.

CASE REPORT

A 39-year-old female was admitted to our Emergency Department due to hypogastric abdominal pain, also with nausea and fever. The patient did not have any family nor personal history of renal disease. Her preoperative work-up included complete blood tests showing absence of alteration in serum creatine, BUN and electrolytes; also, with the presence of leucocytosis with left shift and no other significant findings. According to Hospitals’ Emergency Protocol, abdominal ultrasonography...
was performed supporting the clinical suspicion of AA, no further imaging studies were carried out. Emergency surgical treatment was decided performing a LA. A Foley catheter was placed to monitor both, IAP and urine output (UOP). During intervention gangrenous appendix was observed, also with the presence of important purulent peritoneal fluid in the lower pelvis. The procedure was completed with a pre-established pneumoperitoneum of 12 mmHg (0.016 Bar) and a set air-flow of 3 litres/minute. Overall surgical time was of 45 minutes. During procedure, the patient showed no haemodynamic instability, and maintained an adequate UOP with no transient IAP.

On the first postoperative day, the patient reported persistence of continuous abdominal pain and oliguria (UOP of 300–325 ml/day). Postoperative serial blood test revealed a progressive rise of creatinine levels to a maximum of 5.75 mg/dl; consistent with acute kidney injury (AKI) according to RIFLE criteria [2, 3]. C-reactive protein levels increased to 312 mg/dL with a progressive subsequent decrease. Procalcitonin (PCT) levels, as other clinical septic parameters remained within normality. No correlation was observed between blood tests septic parameters and RF alteration. Surprisingly, despite oliguria and RF impairment no alterations in blood electrolytes were observed. Drug dosage, including antibiotics, required adjustment according to glomerular filtration rate. No nephrotoxic drugs were administered. After conservative treatment with fluid resuscitation and intravenous bicarbonate 1/6 M daily-bolus a complete restoration of RF, also with normalization of UOP, was observed in 48 hours. Control CT-scan showed presence of focal hypodensities in both kidneys, reported as potential renal scars (see Fig. 1). The patient was discharged on Day 16.

**DISCUSSION**

Development of AKI after a laparoscopic procedure has been already reported in the literature, primarily in patients with chronic kidney disease (CKD). Seigneux et al. published a case of a 63-year-old female with known CKD developing exacerbation of RF impairment after robot-assisted surgery [4]. The patient required emergency hemodialysis due to fluid overload, and posteriorly was dependent on renal replacement therapy [4]. Cases of AKI after laparoscopic cholecystectomy have been also reported in literature concerning both patients with CKD [5, 6] and with no previous RF impairment [7, 8], similar to the case we describe.

CO2-pneumoperitoneum is essential for a LA technique. However, it can potentially lead to impairment of cardiovascular, respiratory, renal or even metabolic systems [4–7]. This systemic effect can be attributable to both, induction of IAP and the induced hypercarbia by adsorption of the insufflated CO2 [4, 5, 7, 8]. Ben-David et al. theorized in 1999 that insufflated CO2 may indirectly increase sympathetic autonomic output, stimulating plasma renin-activity and secretion of catecholamines, aldosterone and vasopressin [5, 7]. Main result of this hormonal response is renal vasoconstriction and thus decrease in renal blood flow. However, despite CO2 may have hemodynamic and vascular effects, levels reached in conventional LA are not high enough to be the sole reason of these complications [4].

Hemodynamic effects in laparoscopy are also attributable to IAP. According to international consensus, IAP is defined by a pressure >12 mmHg; abdominal compartment syndrome is considered when IAP >20 mmHg accompanied by organ dysfunction [4, 6, 9, 10]. AKI is one of the first signs in this syndrome [4, 6, 10]. Laparoscopic pneumoperitoneum induced IAP can compromise kidney functionality. IAP produces both, renal parenchyma as well as central venous compression [5–8, 10], and renal arteria vasoconstriction [5–7]; with the subsequent risk of ischemia. Overall, RF deterioration in this setting is speculated to be due to hypoxemia and tubular renal injury [4, 5, 7]. In our reported case, CT-scan showed the presence of focal hypodensities in both kidneys consistent with renal ischemic scars.

Septic etiology such as AA, along with a potential low fluid input intraoperatively and in the immediate postoperative period, may have exacerbated renal failure and could be a confounding factor. However, this is highly unlikely to be the primary etiology. If renal impairment is caused by sepsis, usually produces a prerenal
AKI. Prerenal AKI is accompanied by both blood electrolyte imbalance and azotemia. We did not find any of these in our patient. Despite the possible existence of unknown individual predisposition to the development of this complication, surgeons must consider potential exacerbating factors. The maintenance of norvolemia in conjunction with a low-pressure pneumoperitoneum (Ex. 10–12 mmHg) and brief operative time could minimize the risk of laparoscopy-induced AKI.

A high index of suspicion should be maintained for prompt identification of this condition. Progressive significant rise of serum creatinine, in the absence of a clear cause also with normal blood electrolytes should rise suspicion. Treatment is based on maintenance of adequate fluid balance, removal of nephrotoxic drugs and prevention of AKI related complications. Early nephrology consultation is advised to prevent progressive deterioration of RF and consideration for hemodialysis.

In conclusion, laparoscopy is still a safe surgical approach for abdominal emergency surgery and moreover, AA. Few cases of laparoscopy-induced AKI have been published, probably owing to underestimation of this complication. Surgeons must recall the possibility of this complication, even in young patients.

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None.

CONFLICT OF INTEREST STATEMENT

The authors have no conflicts of interest to declare.

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