

Teaching Point
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The risk of acute kidney injury following laparoscopic surgery in a chronic kidney disease patient

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Introduction

Laparoscopic approaches, particularly when assisted by the Da Vinci robotic interface, are extensively used in urological interventions. Among these interventions, pyeloplasty is one of the most reported. To allow adequate exposure of the operative field, laparoscopic procedures require the creation of a pneumoperitoneum by insufflation of CO₂. This has two corollaries: absorption of CO₂ by the peritoneum and increased abdominal pressure usually monitored and limited to 12–14 mmHg. The induced high intra-abdominal pressure (IAP) and its consequences in modifications of organ perfusion and stimulation of major hormonal systems are associated with functional alteration of various organs. As old patients are more prone to have pre-existing renal dysfunction, these patients are at increased risk of renal complications associated with laparoscopic surgery. In addition, living kidney donors are commonly operated via a laparoscopic approach. The effects of laparoscopy on renal haemodynamics and their potential consequences on kidney function should therefore be acknowledged by nephrologists.

Report of the case

A 63-year-old woman known for chronic kidney disease (CKD) due to recurrent pyelonephritis was diagnosed with a right pyelo-urethral junction syndrome and a robot-assisted laparoscopic pyeloplasty was scheduled. Anaesthesia assessment was performed 2 weeks before surgery. Her glomerular filtration rate (GFR) was estimated at 18 mL/min/1.73m² (CKD Stage V). She had arterial hypertension treated with an angiotensin 2 (AT2) receptor blocker and a thiazide diuretic. Except for haemoglobin of 105 g/L, the other laboratory tests and electrocardiogram as well as the general examination were normal. A general anaesthesia was proposed and the AT2 receptor blocker and thiazide diuretic were stopped 1 day before surgery.

After pre-operative hydration and under non-invasive monitoring, general anaesthesia was induced with propofol and sufentanil orotracheal intubation was performed under muscle relaxation (atracrium). General anaesthesia was maintained with isoflurane and sufentanil. Atracrium was regularly injected to obtain optimal muscle relaxation needed by the laparoscopic surgery. Finally, the patient was positioned on her left side. The haemodynamic profile is described in Figure 1. Soon after generation of the pneumoperitoneum, blood pressure increased, and injections of an alpha-agonist (intravenous clonidine injection) were necessary during the intervention. After 4 h and 15 min of laparoscopy (performed under a constant pneumoperitoneum abdominal pressure between 12 and 15 mmHg), the surgeon had to convert to open surgery because of technical difficulties; 3 h and 30 min were then necessary to finish the operation successfully. During this period, oliguria appeared.

The days after the operation, oliguria did not resolve and creatinine rose substantially. Emergency haemodialysis had to be started because of intractable fluid overload. Renal function did not recover and the patient has been maintained on chronic haemodialysis ever since.

Discussion

Acute tubular necrosis secondary to abdominal hypertension due to the pneumoperitoneum was diagnosed in the reported case. Indeed, we had no alternative diagnosis for the acute renal dysfunction and the patient was at risk given her age, pre-existing CKD and the duration of the pneumoperitoneum.

Few cases of acute kidney injury following laparoscopic surgery are described in the literature [1, 2]. This might be due to underestimation of this entity as many confounding factors are observed in the perioperative period. This case illustrates that laparoscopy has to be included in the factors which can aggravate the loss of renal function in CKD patients. Nephrologists should be concerned by

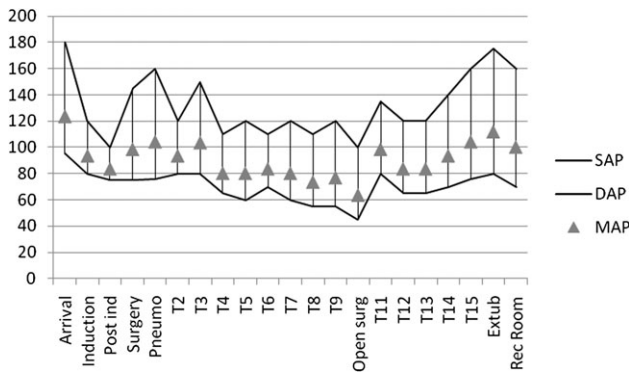


Fig. 1. The haemodynamic profile was recorded throughout the surgical intervention. Non-invasive blood pressure is reported here in millimeter of mercury. SAP: systolic arterial pressure, DAP: diastolic arterial pressure, MAP: mean arterial pressure. T1–T15, period of 30 min after operation start.

renal alterations induced by laparoscopy and surgeons and anaesthesiologists should refer CKD patients (Stages 4 and 5) to nephrologists before the intervention.

Normal abdominal pressure is usually inferior to 5–7 mmHg and equals renal and inferior caval vein pressures. Increased IAP is defined by a pressure >12 mmHg, whereas abdominal compartment syndrome is defined by a sustained abdominal pressure >20 mmHg with associated organ dysfunction [3]. In the abdominal compartment syndrome, acute kidney injury is one of the first sign leading to recognition of the syndrome.

Hypercarbia induced by CO₂ insufflation has hemodynamic and vasodilatory effects, but usually levels reached during laparoscopy do not lead to these complications [4]. Most of the hemodynamic effects of laparoscopy are therefore related to the increased IAP and the associated hormonal modifications. Increased IAP in young adults increases systemic vascular resistance, venal caval pressure, mean arterial pressure and decreases cardiac index, resulting in elevated blood pressure. These haemodynamic effects are proportional to the increased IAP and are dependent on the intravascular volume and on the patient's position suggesting that venous hyperpression and impeded venous return play a major role. The renin–angiotensin–aldosterone (RAA) and sympathetic systems appear stimulated as well as vasopressin release [5–7] and are probably the most important effectors of the increased mean arterial pressure. These haemodynamic effects are usually not significant in healthy individuals but can become more problematic in older patients and in long procedures.

The kidney is the first organ usually affected by a rise in IAP. Demyttanaere *et al.* [8] summarize animal and human studies regarding the effect of elevated pneumoperitoneum on renal blood flow (RBF). Altogether, 14 of 17 studies demonstrate a decrease in RBF under laparoscopic conditions that appears to be dependent on the amount of pressure, on the position and on the volume status, with aggressive fluid repletion correcting part of the decrease in RBF. Changes in renal function also occur under laparoscopy but are more difficult to assess because no reliable marker exist to monitor rapid changes in GFR in clinical practice. Different studies, both in animals and humans have demonstrated

that renal function is altered at least transiently with oliguria and decreased GFR during laparoscopy [8]. One study gave special attention to animals with pre-existing CKD exposed to abdominal insufflations with CO₂ [9] and demonstrates that acute kidney injury may occur in this situation. Interestingly, these effects on systemic haemodynamic and renal function were clearly less apparent when using a retroperitoneal approach. Indeed, insufflation pressure is lower and inferior venal caval pressure does not rise in this condition [10].

Increase in IAP rises renal venous pressure as demonstrated by Bradley [11] in 1947 in healthy men submitted to increased abdominal pressure using a rubber band. Renal venous pressure increased three to four times, whereas renal plasma flow fell strikingly in parallel. In addition, the GFR as measured by inulin clearance also fell during compression in proportion to renal plasma flow. No change was observed in the arterial blood flow and the filtration fraction remained unchanged resulting in a decrease in GFR parallel to the decrease in renal blood flow. In another experiment, corrections of systemic haemodynamics did not correct the decreased RBF and GFR, with a five-time (555%) increase in renal vascular resistance compared to a 30% increase in systemic resistance pointing to renal parenchyma or renal vein compression as the cause of renal dysfunction [12]. Given the fact that blood pressure decreases to maximum 7 mmHg in peritubular capillaries, it can be expected that blood flow will be altered in these vessels if venous pressure and interstitial pressure increase to levels equal or higher. Cortical blood flow was therefore evaluated directly using magnifying endoscopy in the kidney of humans and animals undergoing laparoscopy [13]. From an abdominal pressure of 7 mmHg, pericapillar tubular flow was altered and stopped completely at 25 mmHg, whereas arterial blood flow was unchanged. Damaged tubuli were observed in the subcapsular renal cortex, with features compatible with acute tubular necrosis but no glomerular lesions were observed. Other studies also confirm decreased parenchymal microperfusion pressure in the presence of increased IAP [14].

Living donor nephrectomy under laparoscopic approach is a good model to study the histological effect of IAP as renal biopsy is commonly performed. Shimizu *et al.* [15, 16] reported histological changes in time 0 biopsies in different types of donor nephrectomies. In the subcapsular renal cortex in 37.7% of patients undergoing retroperitoneal laparoscopy and in 46.7% of kidney from patients undergoing transperitoneal laparoscopy, patchy area of tubular necrosis and interstitial haemorrhage in the subcapsular area with congestion of glomerular and peritubular capillaries were observed. The lesions were milder in the retro-peritoneoscopic technique. In comparison, no patient in the open surgery group showed these alterations. In addition, these changes persisted up to 10 months after the nephrectomy in 17% of cases with areas of subcortical necrosis and sclerosis.

In addition to direct increase in venous and parenchymatous pressures, systemic mechanisms may contribute to the observed renal alterations. Activation of the RAA, endothelin [17] and vasopressin [18] systems are documented during laparoscopy and probably play a role in the decreased urine output via their anti-natriuretic and anti-diuretic effects.

Table 1. Preventive measures

Prevention of acute kidney injury during laparoscopy

 Hydration
 Interruption of renin inhibitors, ACE inhibitors and AT2 blockers
 Avoidance of head-up position
 Short intervention time (<3 h)
 Use of lowest intraperitoneal pressure
 Retroperitoneal approach when possible
 Consider open surgery in case of CKD (Stage V)

Their role on the renal dysfunction is less clear as AT2 receptors as well as endothelin blockades appear rather deleterious, suggesting a necessary role of these vasoconstrictive hormones [19].

Volume expansion is the best measure to protect renal function in laparoscopy. This has been demonstrated in different animal and human studies where aggressive volume expansion prevented the decrease in renal blood flow and the decrease in urine output observed during laparoscopy [8, 20, 21]. In addition, the head-up position should be avoided to prevent impeding the venous return further. As the RAA system appeared to be involved in the observed haemodynamic and renal changes, a study has evaluated the effect of angiotensin converting enzyme inhibitors and AT2 blockers on the renal effects of capnoperitoneum in rat [22]. This study demonstrated that urine output, GFR and osmoles excretion were even lower in treated rats compared to controls. ACE inhibitors as well as AT2 blockers should therefore be interrupted in conditions of increased abdominal pressure, such as before laparoscopy, in order to maintain optimal renal perfusion pressure. Finally, the abdominal pressure should be kept as low as possible and duration should be as short as possible in these patients. The retroperitoneal approach should also be preferred (Table 1). In case of acute kidney injury, dialysis and supportive care are recommended, according to the usual management of this condition due to other causes.

This case highlights the fact that kidneys do suffer during laparoscopy. In healthy kidneys, this might be irrelevant to global function, whereas in already damaged kidneys, this additional insult may be sufficient to provoke acute kidney injury. In CKD patients, acute kidney injury is a frequent cause of definitive end-stage renal disease and several risk factors have been identified such as radio-contrast media, other nephrotoxic agents, cardiac failure, cardiac surgery, urinary tract infections, etc. We think that laparoscopic surgery has to be included in this list of factors responsible for deterioration of renal function.

Teaching points

- (1) Laparoscopy results in systemic hypertension.
- (2) The increased abdominal pressure in laparoscopy is sufficient to alter the GFR as well as the peritubular blood flow.
- (3) The duration and intensity of the pneumoperitoneum correlate with the risk of acute kidney injury.

Conflict of interest statement. None declared.

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