CASE REPORT JSLS

Negative Pressure Pulmonary Edema after Laparoscopic Donor Nephrectomy

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ABSTRACT

Introduction: Laparoscopic donor nephrectomy is associated with a 10% chance of morbidity and a 0.03% mortality rate. We present a case of negative pressure pulmonary edema (NPPE) in a healthy subject immediately following a laparoscopic donor nephrectomy. In this report, we will use the case to review the complications of NPPE and to illustrate its management.

Case Description: A healthy 19-y-old male presented at our institution as a living-related donor for left laparoscopic donor nephrectomy. Following the surgery, the patient was reintubated and kept in the intensive care unit secondary to NPPE. The patient experienced an uneventful postoperative course and was subsequently discharged.

Discussion: Although a self-limiting phenomenon, mainstay NPPE therapy requires immediate re-establishment of the airway, adequate oxygenation, and application of positive airway pressure.

Key Words: Negative pressure pulmonary edema, Laparoscopic nephrectomy, Donor nephrectomy, Laparoscopic surgery morbidity.

INTRODUCTION

Laparoscopic donor nephrectomy is associated with a 10% chance of morbidity and a 0.03% mortality rate.[1,2] Two cases of unilateral negative pressure pulmonary edema (NPPE) have been previously described in the literature.[3] We report a case of NPPE in a healthy subject immediately following an uneventful laparoscopic donor nephrectomy; a review of NPPE and its management is also presented.

CASE REPORT

A healthy 19-y-old male presented to our institution as a living-related donor for left laparoscopic donor nephrectomy. Other than a body mass index of 31.4, his examination and history were unremarkable. After premedication with 2 g midazolam, general anesthesia was induced with 150 mg propofol, 150 mcg fentanyl, and 9 mg vecuronium, and endotracheal intubation was performed atraumatically. The patient was maintained with desflurane and nitrous oxide, and placed in the lateral decubitus position. The nephrectomy lasted 3.5 h and was uneventful. The patient received 6 L of crystalloids and 25 g mannitol. Estimated blood loss was 50 mL and urine output was 200 mL.

Following the procedure, the patient was returned to the supine position and reversed with 5 mg neostigmine and 0.80 mg glycopyrrolate. Once he was responsive with adequate respiratory and neuromuscular function, the patient was extubated. Immediately afterwards, the patient became agitated and made vigorous inspiratory efforts. His pulse spiked to 130, his blood pressure rose to 165/100 mm Hg, and his oxygen saturation dropped to 88%. Continuous positive airway pressure with 100% oxygen was applied by using a face mask. Coarse inspiratory rhonchi were heard bilaterally on auscultation, and copious, pink, frothy sputum was obtained with suctioning. Flash pulmonary edema was suspected, and the patient was reinduced with 50 mg propofol and 120 mg succinylcholine and reintubated atraumatically. Blood gas demonstrated a pH of 7.10, $PaCO_2$ of 84, PaO_2 of 46, and a bicarbonate level of 29. Initial portable chest radiograph showed bilateral

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patchy opacities and interstitial infiltrates. Echocardiogram demonstrated normal sinus rhythm with sinus tachycardia. Oxygen saturation and blood gas parameters normalized on 10cm H_2O positive end-expiratory pressure (PEEP) and 100% FiO_2.

Postoperatively, the patient was transferred to the intensive care unit, where he was maintained on a 60% FiO_2 and a PEEP of 20. The patient was diuresed with furosemide and his urine output increased to 2500 mL over the next few hours. A transthoracic echocardiogram demonstrated normal cardiac function. By postoperative day 1, the patient's chest X-ray, respiratory parameters, and blood gas parameters had improved, and he was successfully extubated. Over the following 3 d, the patient experienced an uneventful postoperative course and was discharged to home on postoperative day 4. Two weeks later, patient had no complaints at his postoperative follow-up visit.

DISCUSSION

We present a case of an otherwise healthy 19-y-old who underwent laparoscopic donor nephrectomy complicated by NPPE, a well-described, but uncommon phenomenon that involves the development of pulmonary edema secondary to an acute upper airway obstruction. NPPE is characterized by the formation of negative intrapleural pressure, which is transmitted to the pulmonary interstitium, which then increases the venous return to the right side of the heart with a concomitant increase in pulmonary arterial and pulmonary capillary hydrostatic pressures. The increased venous return also results in the distention of the right ventricle, leading to interventricular septal shift and a resultant reduction in left ventricular compliance and cardiac output.[4] Furthermore, the increased pulmonary capillary hydrostatic pressure causes an increased transcapillary pressure gradient, favoring the transudation of fluid into the interstitial space.[5,6] This capillary leak results in alveolar edema and the observed serosanguinous, pink, frothy fluid, a classic finding in NPPE.[6]

Additionally, the lateral decubitus position can alter the physiology of pulmonary ventilation and perfusion and can consequently also contribute to pulmonary edema. The dependent zones of the lung become hyperperfused and hypoventilated, whereas the nondependent portion becomes hypoperfused and hyperventilated, thus creating a ventilation/perfusion mismatch. The imbalances consequently increase the propensity for fluid extravasation in the dependent portion of the lungs (the "down lung syndrome") and can result in pulmonary edema.[7] Furthermore, prolonged pneumoperitoneum and high intraabdominal pressure during LLDN can compress the renal arteries causing decreased renal blood flow and oliguria.[3] This would explain our patient's low intraoperative urine output of 200 mL. The resuscitative fluid may have also worsened the fluid extravasation into the lungs and capillary leak caused by NPPE.

In one of the first large reviews of NPPE, Deepika et al.[5] reported an incidence of 0.094% and described a typical onset within minutes of airway obstruction. This obstruction was more common in the postextubation period (73.4%) and was likely due to laryngospasm.[5] In the remaining 26.6%, acute upper airway obstruction occurred during preoperative management of the airway due to tumors, Ludwig's angina, and laryngospasm.[5] Of those with NPPE, 11% were young, healthy patients; this was likely due to their ability to generate profound negative intrathoracic pressure.[5]

Prevention of NPPE necessitates close monitoring of perioperative fluid administration and urine output. In many patients, adequate preoperative hydration can reduce the need for large iatrogenic fluid boluses.[3] A high level of vigilance must also be maintained in all patients at risk for airway obstruction. In general, NPPE is a self-limiting phenomenon, with radiologic clearing and normalization of arterial blood gas parameters typically within 48 h. Until the condition resolves itself, the aim of therapy is supportive. Most patients require reintubation and ventilation with positive airway pressure to ensure adequate oxygenation.[5]

CONCLUSION

Our case of a young, healthy male who underwent laparoscopic live donor nephrectomy demonstrates the unexpected development of NPPE, a relatively rare event with a high degree of morbidity. The present case emphasizes the importance of constant vigilance and early diagnosis. Immediate re-establishment of the airway, adequate oxygenation, and application of positive airway pressure represent the cornerstones of the therapy.

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