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Case Report

Hot Peritoneal Lavage Fluid as a Possible Cause of Vasovagal Reflex During Two Different Surgeries for Bladder Repair in a Foal

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ABSTRACT

A 72-hour-old Arabian filly was presented with respiratory distress and abdominal distension. Diagnostic ultrasound identified free peritoneal fluid and a collapsed bladder leading to a diagnosis of uroperitoneum. Electrolyte abnormalities of K^+, Na^+, and Cl^-; hypovolemia; high blood urea nitrogen; and creatinine prompted a first stabilization before surgical approach. The laparotomy under general anesthesia provided drainage of the urine and allowed the surgical correction of the ruptured bladder. Two surgeries were required to repair the bladder, and in both occasions, sudden profound hypotension, bradycardia, and third-degree AV-block appeared during peritoneal lavage with microwave-heated fluids. A normal sinus rhythm and blood pressure were restored with atropine and concomitant inotropes administration as in a vasovagal response. The filly recovered uneventfully from anesthesia. If not properly anticipated, recognized, and treated, foals with preexisting electrolyte abnormalities are at greater risk of hemodynamic instability. This can have many causes, but this report describes a possible concomitant detrimental trigger which may have contributed to the cardiovascular side effects. Post hoc testing of the fluid warming process with the same settings previously used during both surgeries revealed fluid temperatures of >41.7°C. A peritoneal lavage with excessively microwave-heated fluids might have induced a sudden vasodilation leading to a vasovagal reflex. It is advisable to verify the temperature of the lavage fluid when using quick warming devices, as microwave, and fluids at ideally slightly above the normal body temperature are indicated to perform safe peritoneal lavage.

1. Introduction

Uroabdomen is a common condition in neonate foals with a greater occurrence in males, and the most frequent cause is leakage from a bladder defect. Hypovolemia, hyponatremia, hypochloremia, hyperkalemia, azotemia, and metabolic acidosis are reported as concurrent problems and most often occur in foals that suckle the mare's milk which is further relatively high in K^+ and low in Na^+. Respiratory distress is also reported and attributed to compression of the thorax by the uroabdomen, limiting its normal excursions, although fluid can also accumulate directly in the pleural space as described in the present report and elsewhere [1,2] possibly leading to hypoxia and hypercapnia. Clinical signs of uroperitoneum typically include tachycardia, tachypnea, straining to urinate, and variable mentation status, according to azotemia and hyponatremia, and the immediate goals of treatment include cardiovascular and respiratory improvement by

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slow drainage of the abdomen and electrolyte derangement correction. The increase of intraabdominal pressure has been implicated in the evolution of abdominal compartment syndrome [3] leading to a further exacerbation of cardiovascular and respiratory status such as reduced cardiac output and pulmonary compliance [4]. Correction of hyperkalemia to a serum concentration <5.5 mmol/L is of paramount importance before general anesthesia to prevent cardiac arrhythmias and emergencies [5]. As reported in human literature, abdominal decompression may precipitate adverse physiologic and metabolic events that is a large increase in pulmonary compliance and the risk of reexpansion pulmonary edema, especially if large airway pressure was instituted to sustain ventilation [6], a sudden drop of systemic vascular resistance, as well as the washout of toxic products accumulated within the tissues, which would rapidly return systemically [7]. Along with the concurrent hypoxia, anaerobic metabolism induces the accumulation of lactic acid as well as adenosine and potassium that, once circulation is restored, would lead to arrhythmia, myocardial depression, and further vasodilatation [8]. If not properly anticipated, recognized, and treated, foals with preexisting electrolyte abnormalities are at greater risk of such cardiac complications. This report describes a possible concomitant detrimental trigger which may have contributed to the cardiovascular side effects such as a peritoneal lavage with highly microwave-heated fluids which might have induced a sudden vasodilation leading to a vasovagal reflex.

2. Case Description

A 72-hour-old, 50-kg Arabian filly was referred to the Veterinary Teaching Hospital for a suspected uroperitoneum. At admission, the filly was ambulatory, mildly depressed but regularly nursing. It presented with fever (39.2°C), respiratory rate (RR) 40 breaths/min with flared nostrils. The heart rate (HR) was 110 beats/min, mucous membranes were pink with a capillary refill time of 2.5 seconds, bowel sounds were decreased, and the abdomen was distended. Blood analysis revealed packed cell volume 41%, total protein 7 mg/dL, elevated blood urea nitrogen 42.6 mg/dL (reference range [r.r.], 10–20) and creatinine 3.7 mg/dL (r.r. up to 1.3), pH 7.36 (r.r., 7.32–7.44), arterial partial pressure of carbon dioxide ($PaCO_2$) 6.72 kPa (r.r., 6.53–7.47), hyperkalemia 5.9 mmol/L (r.r., 1.9–4.1), hyponatremia 114 mmol/L (r.r., 128–142), and hypochloremia 82 mmol/L (r.r., 100–111). Abdominal ultrasound revealed an empty bladder with a tear of the wall, abundant peritoneal fluid, and free fluid in the thorax. Abdominocentesis was not performed, but with a diagnosis of ruptured bladder and subsequent uroperitoneum, an urgent surgical treatment was scheduled after that the severe electrolyte derangements were addressed, even if not thoroughly corrected.

IV fluid therapy started with 1 L 0.9% NaCl administered as bolus before antimicrobials (cefquinome 1 mg/kg IV—Cobactan 4.5%, Gellini, Italy) and nonsteroidal anti-inflammatory therapy (meloxicam 0.6 mg/kg IV—Metacam, Boehringer, Italy).

The foal was premedicated with 0.1 mg/kg diazepam (Diazepam, Hospira, Italy) and 0.1 mg/kg butorphanol (Nargesic, ACME, Italy) IV. Five minutes later anesthesia was induced with 2.2 mg/kg ketamine (Ketavet 100, Intervet, Italy) IV. The trachea was intubated, and the filly was placed in dorsal recumbency on an electric heating pad in a slight antitrendelenburg position. The endotracheal tube was connected to a circle breathing system of a small animal anesthesia machine. Anesthesia was maintained with a mean ± standard deviation (SD) expired sevoflurane (SevoFlo, Esteve, Italy) fraction (FE'Sevo) of 2 ± 0.2% in 100% oxygen, and 0.9% NaCl was infused at 2.5–8 mL/kg/hr.

A Mindray BeneView T5 machine was used to monitor the electrocardiogram trace and HR, end-tidal carbon dioxide concentration, FE'Sevo, RR, hemoglobin oxygen saturation (SpO_2), esophageal temperature (T°), and blood pressure, measured directly from the facial artery with a 22-gauge catheter connected to a calibrated transducer zeroed and positioned at the heart level (manubrium) throughout the procedure. A continuous rate infusion (CRI) of dobutamine (Dobutamina, Bioindustria LIM, Italy) was started using incremental doses of 0.25 μg/kg/min as described elsewhere [9] to maintain mean arterial blood pressure (MAP) ≥60 mm Hg. A laparotomy, which started 30 minutes after induction of anesthesia, allowed slow abdominal drainage. Twenty minutes later an arterial blood gas (ABG) showed insufficient oxygenation (PaO_2 10.39 kPa, SaO_2 88%), respiratory acidosis (pH 7.12, $PaCO_2$ 11.41 kPa, HCO_3- 27.7 mmol/L), and a worsening hyperkalemia (K^+ 6.5 mmol/L). Until now, mean ± SD SpO_2 and body T° were 89 ± 9.6% and 37.7 ± 0.2°C, respectively. The IV fluid therapy was complemented with 5% glucose (6 mL/kg/hr), and assisted ventilation was provided manually. A loading dose of 5 μg/kg fentanyl (Fentanest, Pfizer, Italy) was administered IV over 2 minutes to improve analgesia. After 45 minutes of surgery at the conclusion of the bladder repair and before abdomen closure, the surgeon ordered a warm peritoneal lavage with 5-L microwave-heated sterile saline at 1,000 watt for 12 minutes. At intraabdominal application of about 3 L of warm fluid, immediately severe hypotension (MAP dropped from 64 to 36 mm Hg), severe bradycardia (HR dropped from 89 to 53 beats/minute), and third-degree A-V block developed (Fig. 1). Sevoflurane administration was discontinued and manual ventilation maintained at a rate of 10 to 12 breaths/min. The hemodynamic instability was addressed with two doses of IV atropine (0.01 mg/kg) (Atropina solfato, Ind. Farm. Galenica Senese, Italy) administered with a 2-minute interval while dobutamine was running at 0.5 μg/kg/min. Surgery was completed in further 15 minutes and ABG at that time resulted in pH 7.24, $PaCO_2$ 7.55 kPa, PaO_2 11.73 kPa, SaO_2 96%, HCO_3- 22.3 mmol/L, base excess (BE) −5 mmol/L, persistent hyponatremia (116 mmol/L), and hyperkalemia (6.1 mmol/L). By that time, HR and MAP had returned to normal values. The filly recovered uneventfully from anesthesia and was consciously suckling from the mare within 1 hour. In the following hours, the foal was maintained on 0.9% NaCl with 5% glucose (100 mL of glucose 50% in 900-mL saline) at 5 mL/kg/hr to address electrolyte abnormalities.

Ten hours later, the foal was depressed, not suckling, and showed a distended abdomen with stranguria.