Urinary bladder exstrophy–epispadias complex (EEC) is a rare congenital genitourinary malformation (prevalence 1/10,000) and is characterized by an evaginated bladder plate, epispadias, and an anterior defect of the pelvis (pubic diastasis), pelvic floor abdominal wall, and spine (7% anomalies).\(^1\) A multidisciplinary team consisting of surgeons, anaesthesiologists, paediatricians, nutritionists, pharmacologists, nurses, and child life specialists are essential for a better outcome. Intraoperative anaesthesiologist challenges include opioid-based respiratory depression, hemodynamic instability due to blood loss because of pelvic osteotomies in older children, increase fluid loss from exposed bowel in bladder augmentations, inadequate assessment of volume status (no urine output measure), overhydration resulting in flooding of the surgical field by patient’s urine, severe postoperative pain because pelvic external fixator in situ and lower extremity traction, and long duration of surgery.

Here, we report a clinical situation of 1.5-year-old male child who presented with exstrophy of bladder with epispadias along with developmental dysplasia of the hip. Surgery was planned for functional closure of the bladder with bilateral Salter’s osteotomy and external fixation. General anesthesia with a caudal epidural block was given. Written and informed consent was taken for publication. The whole intraoperative period was uneventful, and the patient’s trachea was extubated successfully. In the postoperative period, we detected sudden bradycardia with associated oxygen desaturation 10 minutes after shifting from the operating room. For the management of bradycardia, the patient’s trachea was reintubated and underwent three cycles of cardiopulmonary resuscitation.

On arterial blood gas (ABG) examination, severe metabolic acidosis (pH 7.205, pCO\(_2\) 43.3 mmHg, pO\(_2\) 306.8 mmHg, base deficit 10.9 mmol/L, lactate 4.7 mmol/L) was detected. Acidosis was corrected by infusing a mixed solution of sodium bicarbonate (8.4%) and dextrose 10% (1:1 ratio) along with proper hydration to the patient. The patient’s trachea was extubated successfully after correction of acidosis and overnight mechanical ventilation. The possible differential diagnosis made by the anaesthesiologist team includes hypoxia, hypovolemia, acidosis, electrolyte abnormalities, opioid overdose, and bladder traction. We ruled out hypoxia as a cause of bradycardia because bradycardia started before oxygen desaturation. Normal respiratory drive ruled out opioid overdose. The diagnosis of metabolic acidosis as a cause of bradycardia was made following the ABG report.

Metabolic acidosis is common in patients undergoing extensive surgery involving significant blood loss.\(^2\) This might be due to a combination of blood loss and electrolyte disturbances following the administration of large amounts of crystalloid, colloid, and blood products. The base deficit, an indicator of metabolic acidosis, correlated directly with the severity of hemorrhagic shock and can be used in quantifying the response to hemorrhage in both traumatic and nontraumatic patients.\(^3\) The maximum base deficit was recorded after the end of surgery.\(^4,5\) In our case, at the end of the surgery, base deficit detected was 10.9 mmol/L. The cause of acidosis might be due to inadequate hydration because of poor assessment of volume status during the intraoperative period, underestimated actual blood loss (disappears into the pelvic tissues), blood transfusion related, long duration of surgery, and poor acid clearance because of pediatric age-group, or a combination of the above. During the intraoperative period, we advocate judicious use of intravenous fluids with CVP (central venous pressure) monitoring, invasive blood pressure monitoring, regular monitoring of pH, base deficit, hemoglobin, and electrolytes in patients at-risk for blood loss (osteotomies) and multimodal approach to analgesia. Meticulous attention to fluid intake and output should is also required.

**Declaration of patient consent**

Taken from the patient.

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Nil.

**Conflicts of interest**

There are no conflicts of interest.

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To the Editor,

The severity of respiratory illness in COVID-19 depends on the imbalance between the pro-inflammatory and anti-inflammatory mediators in the lung parenchyma. The phases of the illness have been divided into the early respiratory tract infection followed by acute inflammation. The third phase is variable depending on patient characteristics i.e. presence of co-morbidities, viral load exposed, baseline immunity, the initiation of early treatment. There could be either resolution of the illness or severe illness.

Severe illness leads to cytokine storm (IL-6, TNF-α), leaky capillaries, hypoxic respiratory failure, multiorgan involvement leading to adult respiratory distress syndrome (ARDS) and subsequently invasive or non-invasive ventilation, shock all of which contributes to significant morbidity, and subsequent mortality. The severe illness also leads to a hypercoagulable state which is evaluated using d-dimer and fibrin degradation products and managed with heparin (unfractionated or low molecular weight). TNF-α released during cytokine storm causes endothelial damage which leads to thrombosis. The thromboembolic events occurring thereafter contribute to significant morbidity and mortality.

Cytokines like IL-6, TNF-α eventually leads to the release of reactive oxygen species (ROS) and reactive nitrogen species from the mitochondria which is responsible for end-organ damage, thrombosis, and shock [Figure 1].

Presence of comorbidities like hypertension, diabetes mellitus, ischemic heart disease, respiratory ailments, chronic kidney disease, obesity and the elderly age groups are the ones with a low baseline immunity and thus are easily susceptible to develop serious illness once COVID-19 positive.

The human body is naturally equipped with enzymatic and non-enzymatic antioxidant systems to prevent damage to cells, and vital organs due to free radicals. Glutathione peroxidase, catalase and superoxide dismutase are the enzymatic antioxidants. The non-enzymatic antioxidants are vitamin E, vitamin C, thiol antioxidants (Glutathione, Thioredoxin and lipoic acid), melatonin, carotenoids, and natural flavonoids. In vivo, scavenging of ROS is predominantly mediated by Glutathione and glutathione peroxidase which is its regulator enzyme. When production of ROS is uncontrolled there is depletion of glutathione as a result of which the patient gets susceptible to immunosuppression, organ damage, increased vascular...