



Case Report

Anaesthetic management of a patient with glucose-6-phosphate dehydrogenase deficiency undergoing robotic-assisted laparoscopic radical prostatectomy

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Abstract

Glucose-6-phosphate dehydrogenase deficient patients may develop acute haemolysis after exposure to the oxidative stress of surgery, and certain anaesthetic or analgesic agents. The steep Trendelenburg position, pneumoperitoneum, and associated extreme hemodynamic changes in robotic surgery add on to oxidative stress. Here, we present a 68-year-old Glucose-6-phosphate dehydrogenase deficient patient who uneventfully underwent robotic-assisted laparoscopic radical prostatectomy focussing on avoidance of drugs predisposing haemolysis, minimising surgical stress, providing adequate analgesia, stable haemodynamics and depth of anaesthesia that is unique to robotic surgery.

Keywords: bispectral index monitor; epidural analgesia; glucosephosphate dehydrogenase deficiency; oxidative stress; robotic surgical procedure.



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Introduction

Glucose-6-phosphate dehydrogenase (G-6-PD) deficiency is an X-linked recessive genetic disorder caused by insufficiency of the enzyme in the hexose-mono-phosphate (HMP) shunt pathway resulting in decreased production of nicotinamide adenine dinucleotide

phosphate hydrogenase (NADPH). NADPH protects red blood cells (RBCs) from oxidative stress, and its deficiency makes red blood cells susceptible to haemolysis and renal failure.¹ Haemolysis becomes evident 1-3 days following the oxidative stress and usually resolves itself occasionally requiring blood transfusion.² Administering anaesthesia

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for G-6PD patient pose unique challenges which have been highlighted in this case report.

Case report

A 68-year-old male patient weighing 82 kilogrammes admitted with prostate cancer. The patient had G-6-PD deficiency (Class III variant) since childhood. There was a history of haemolysis, five years back, post chemotherapy leading to drop in haemoglobin to two gm% requiring blood transfusions. Presently patient was planned for robotic-assisted laparoscopic radical prostatectomy (RALRP). Preoperative investigations revealed haemoglobin-9 gm%, unconjugated bilirubin-0.75 mg/dl and reticulocyte count (RC)-1%, LDH-160 U/L, PT/INR-10.10 seconds/0.99. We assured the availability of cross-matched blood. The patient was premedicated orally with granisetron 2 mg and alprazolam 0.25 mg. Intravenous ceftazidime 1 gm + tazobactam 125 mg were given preoperatively. Inside operation theatre, an intravenous line was secured and an epidural catheter was placed between L1-L2 intervertebral spaces. After preoxygenation, induction of anaesthesia was performed with fentanyl 2 µg/kg, propofol 2.5 mg/kg, intubation was facilitated with atracurium 0.5 mg/kg using video laryngoscope. Epidurally 12 ml of Bupivacaine (0.25%) was given. Pneumoperitoneum was created with intraabdominal pressures of 12 mmHg and patient was positioned in steep Trendelenburg position (45°). da Vinci robot was attached to robotic arms. Apart from standard monitoring, end tidal CO₂ (ETCO₂), peripheral nerve stimulator (PNS), bispectral index (BIS), arterial blood gas (ABG), random blood sugar (RBS), temperature and urine output were also used.

Balanced anaesthesia was provided with oxygen-air mixture 50:50, desflurane (up to MAC 0.5), propofol infusion and PNS guided atracurium infusion. A total of 200 µg fentanyl was given over a period of three hours. Epidural bupivacaine (0.25%) was repeated intermittently. BIS was maintained between 40 and 60. Minimal intravenous fluid (plasmalyte-A: 700 ml) was given. Blood loss and urine output were 100 and 350ml respectively. Lactate levels in ABG and RBS remained normal. The patient maintained stable haemodynamics throughout the surgery.

On completion of surgery, the trachea was extubated after reversal of neuromuscular blockade.

In postoperative period patient received an infusion of fentanyl 1-2 µg/kg/hr along with epidural Bupivacaine (0.125%) for analgesia. On 2nd postoperative day haemoglobin dropped to 7.7 gm%, RC was 3.5% and Heinz body was not seen, LDH was 170 U/L and indirect bilirubin 1.25 mg/dl. One unit of packed red blood cells was transfused. The patient was discharged on the 4th postoperative day with a haemoglobin of 8.6 gm%.

Discussion

G-6-PD deficiency is the most common enzyme deficiency disorder worldwide.³ Oxidative stress, various drugs and

infections are known to precipitate haemolysis in G-6-PD deficient patients resulting in acute haemolytic anaemia.¹

Acute intravascular haemolysis becomes evident 2-3 days following exposure and recovery is marked by reticulocytosis. Laboratory findings may include anaemia, reticulocytosis, decreased serum haptoglobin and elevated indirect bilirubin and LDH. In our patient haemoglobin level dropped to 7.7 gm%, reticulocyte count increased to 3.5%, indirect bilirubin 1.2 mg/dl, LDH-170u/l but no Heinz bodies in peripheral smear suggesting mild haemolysis. Our anaesthetic management focused on avoiding oxidative stress and the drugs implicated in haemolysis and monitoring of perioperative haemolysis.

Pneumoperitoneum during RALRP is known to increase oxidative stress by increasing ischemia-modified albumin.⁴ Increased intraabdominal pressure may cause splanchnic ischemia followed by reperfusion due to deflation. That leads to increased vascular permeability and production of oxidative radicals by neutrophils and inflammatory cells.⁵ During active infections, oxygen free radicals are produced either by inflammatory neutrophils or by use of antimicrobials and may precipitate haemolysis during the perioperative period. Therefore adequate treatment of active infections and administering an appropriate antibiotic (ceftazidime) which does not precipitate perioperative haemolysis is of extreme importance. Right from tracheal intubation using video laryngoscope and epidural analgesia before pneumoperitoneum helped in preventing haemodynamic stress response. We provided liberal analgesia and adequate depth of anaesthesia to avoid acute swings in haemodynamics. Bupivacaine was used and not lignocaine in epidural test dose because lignocaine can precipitate haemolysis in G6PD deficient patients.⁶ Fluid management in our patient was according to robotic surgery fluid guidelines. Active warming measures were taken to maintain normothermia. Normal blood pH and proper sugar control were assured to prevent haemolysis.⁷

Enzyme (G-6-PD) levels are also low in platelets.⁸ Hence coagulation parameters are necessary for preoperative investigations especially when the neuraxial block is planned. Haemolysis in G-6PD deficient patients is influenced by the type and dose of the offending drug. Petz and others⁹ listed a group of drugs, known to cause haemolytic anaemia. That include antimicrobials sulfonamides, nitrofurantoin and chloramphenicol, non-steroidal anti-inflammatory drugs (NSAIDs), anticonvulsants, diuretics, insulin, ranitidine and thiopentone.¹⁰

Acute haemolysis in the intraoperative period should be strictly looked for by colour of urine as the immediate signs are typically masked during anaesthesia. Once diagnosed, discontinuation of the offending agent and maintenance of urine output by infusion of crystalloids along with diuretics has to be ensured. Postoperatively, complete blood count on a daily basis should be followed to monitor

the need for blood transfusion.⁹ Our patient had mild haemolysis evident on 2nd postoperative day reflected by a drop in haemoglobin level to 7.7 gm% and an increase in RC from 1% to 2.5%. It was corrected by transfusing one unit packed cells.

To conclude, G-6-PD deficiency poses a risk of life-threatening haemolysis which can lead to anuria and acute renal failure. Robotic surgery adds challenges in a G-6-PD deficient patient due to its steep Trendelenburg positioning and marked haemodynamic variability. Hence providing adequate analgesia, depth of anaesthesia, analgesia and stable haemodynamics and avoiding haemolysis precipitating drugs are key to success.

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