Anesthetic management in ABO incompatible kidney transplant

Sir,

With rising demand of kidney transplant and limited availability of ABO-compatible kidney donors, ABO incompatible (ABOi) kidney transplant is a viable option. The median waiting time depended on the blood type of patients, but it is reported to be around 4 years for all patients on the OPTN report.[1] With the current understanding of immunological factors and the availability of drugs such as rituximab and procedures such as plasmapheresis, immunoabsorbent techniques the graft survival of incompatible transplanted kidney is comparable with the compatible one.[2,3]

End-stage renal disease (ESRD) patients suffer with many comorbidities such as anemia, platelet dysfunction, ischemic cardiomyopathy, dyselectrolytemia, uremia and along with ABOi, the anesthetic management tends to play a vital role in successful transplant and graft survival.[4]

Here, I am describing the methods used in our set up for such cases. A 34-year-old male with ESRD was on regular hemodialysis twice a week. His blood group was A +ve. He was nonhypertensive with heart rate of 76/min. His blood urea was 152 mg/dl, serum creatinine - 9.93 mg/dl, serum Na - 139 mEq/L, and serum K - 5.33 mEq/L. Anesthetic examination and other investigations including two-dimensional echocardiography were within normal limit. Whereas the patient's blood group was A +ve, the donor was patient's relative whose blood group was B +ve. She was a known hypertensive lady controlled on amlodipine 5 mg. No other comorbidity was present in the donor, and all her investigations were within normal limits.

Initially, the recipient's anti-B titer was 1:64. To reduce the anti-B titer to <1:8 regular plasmapheresis was added to the dialysis schedule. Two weeks before KT, rituximab was administered, and consequently, the titer was decreased up to 1:16, 7 days before surgery, so further plasmapheresis was planned with concomitant addition of fresh-frozen plasma (FFP) to obviate deficiency of coagulation factors. The patient was started on tablet Pangraf 2.5 mg BD; tablet Myphens 36 mg BD for immunosuppression 3 days prior to KT. Injection Solu-Medrol 250 mg and injection thymoglobulin 50 mg were given on the day of KT. Hemodialysis and plasmapheresis were performed on alternate days until the evening before the operation.

After attaching all the monitors, a dedicated intravenous access and an arterial line were secured. Before induction of anesthesia right internal jugular vein was cannulated with triple lumen intravenous catheter under ultrasound guidance. The patient had a central venous pressure (CVP) of 4 cm of water and blood pressure of 160/70 mmHg. Arterial blood gas was showing a pH of 7.42 and serum K^+ of 3.2 mmol/L. Leukodepleted blood of Group A +ve was cross-matched and saved along with 4 units of AB blood group FFP.

General anesthesia was induced with injection fentanyl 2 mcg/kg, midazolam 1 mg, and titrated dose propofol. Orotracheal intubation was facilitated with 30 mg of atracurium. Patients were monitored using electrocardiogram, intra arterial blood pressure, CVP, pulse oximetry, and nasopharyngeal temperature. The temperature was maintained using warm air blanket. A very limited fluid was given to the patient till the end of the renal vein anastomosis maintaining a CVP between 4 and 7 cm of water. Minutes before unclamping of graft artery CVP was gradually raised using crystalloid solution mostly normal saline up to 15 cm of water. Injection Solu-Medrol 250 mg was also repeated intraoperatively. Total blood loss remained under allowable limits, so blood transfusion was avoided. After successful vascular graft, the transplanted kidney was observed for signs of turgidity, hemostasis, vascular sufficiency, function, and rejection. The intravenous fluid was titrated to keep the CVP between 12 and 15 cm of water because the transplanted kidney was pouring lot of urine which may be due to ineffective early concentrating function of the kidney.

The patient was nursed in dedicated kidney transplant unit with barrier nursing care along with strict asepsis. Other than routine postoperative care which includes pain control, core temperature maintenance, check on bleeding, and postoperative nausea and vomiting one needs to monitor and preserve kidney function and its acceptance by the host body, respectively. The measures taken in this path were input – output chart, daily KFT, Hb level, anti-B titer and continuation of immunosuppressant and steroids. In our case, need for hemodialysis did not arise although patient necessitated two units of pack cell transfusion of Group A +ve blood.

For a successful ABOi KT, prophylactic measures to prevent acute rejection are effective suppression and monitoring of immune system. The role of plasmapheresis, immunosuppressants, rituximab, and Intravenous immunoglobulin (IVIG) are vital.[5] Both plasmapheresis and IVIG have an effect on reducing the circulating antibody. The use of rituximab lies in the idea of slowing the growth and production of antibody by immature B cells, as a result, further anti-B titer levels will be kept under control in the future.[6]

The prevention of infection is a major concern in these patients. Such patients who are heavily immunosuppressed do not produce an inflammatory response such as fever, raised leukocyte count to infection and are very prone to bacterial, viral and fungal infections immediate postoperatively.[7] This proves the value of minimal invasiveness during perioperative period.

The choice of blood products in our patient are leukodepleted PRBC of A +ve group and FFP of AB group. Whereas the best possible situation is when blood transfusion is avoided but if needed perioperatively choices lie with us to balance the need of the blood and avoid any autoimmune reaction. The plasma of recipient contains a minimal amount of anti-B antibody titer when grafting is done therefore the best choice is to use FFP of AB group because it virtually eliminates the risk of any antigen–antibody reaction.

A tight balance is to be maintained in administering intraoperative fluid to the patient as there are no means of fluid removal from the body. Fluid given before anastomosis and graft function usually spreads into the interstitial space and results in edema formation. The reduced central compartment post dialysis and low albumin level summons the use of low dose of induction agents although high cardiac output states causes slow rise of effective cerebral pressure of inhalational agents.

Postoperatively, the immune system is kept suppressed till the accommodation of kidney into host body develops. To help accommodate kidney, the preoperative immunosuppressive regimen is continued and titrated into the postoperative period, and a daily check of anti-B titers and kidney function is performed.

Acknowledgment

Author would like to thank the hospital and the staff.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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Access this article online	
Website: www.saudija.org	**Quick Response Code**
DOI: 10.4103/sja.SJA_34_17	

How to cite this article: Gupta A, Bhan S. Anesthetic management in ABO incompatible kidney transplant. Saudi J Anaesth 2017;11:352-4.