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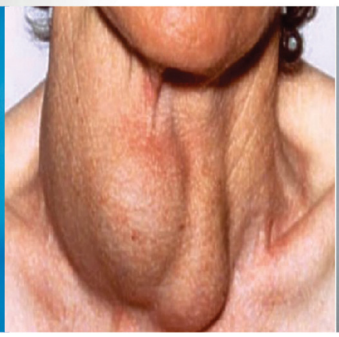
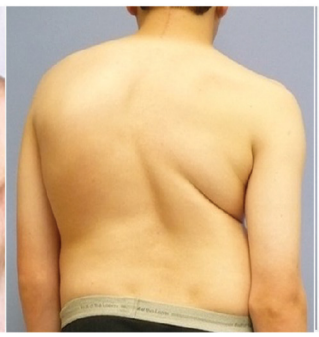
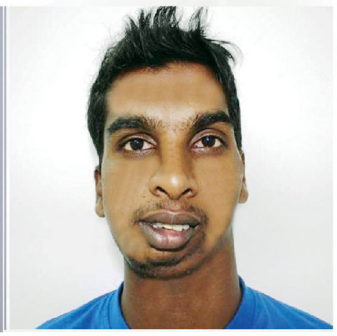
# Case Discussion on Anesthesiology

Tulsi Nag

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Chapter

# 4

# A Kidney Transplanted Patient Put for Nontransplant Surgery

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**Q. What is the purpose of organ transplantation?**

Organ transplantation is the treatment of choice for end stage organ failure from any of the wide variety of causes.

**Q. What is meant by transplantation? How is it categorized?**

Transplantation is the act of transferring organ, tissue or cell from one site to another. Transplantations are divided into three categories:

1. *Autotransplants:* Transplant of organ or tissue from one part of an individual to other part of the same individual. (Skin graft, vein graft for CABG). No immunosuppression is required.
2. *Allotransplant:* Transfer of solid organ from one individual to another individual of same species. Immunosuppression of the recipient is required to prevent graft rejection.
3. *Xenotransplants:* Involves transfer across species, barriers. Graft is taken from other species. Relegated to laboratory. Complex and potent immunological barrier needed.

**Q. Why graft rejection occurs?**

Graft rejection is due to an immune response in the host to foreign alloantigen after organ transplant from genetically non-identical donors.

It is a complex process involving T lymphocytes, B lymphocytes, macrophages, and cytokines with resultant local inflammatory injury and graft damage.

In humans, the main antigen involved in triggering rejection is the human leukocyte antigen HLA system. The HLA system initiates rejection and graft damage through humoral or cellular mechanisms. Cellular rejection is more common after organ transplants and mediated by T lymphocytes. Graft rejection reactions have various time courses:

- *Hyperacute:* Within minutes after transplanted organ is reperfused.
- *Acute:* Most common and occurs within days to a few months post-transplant, becoming less with moderate immunosuppression.
- *Chronic:* Occurs within months to years post-transplant.

## Q. What specific measures are followed during laparoscopic procedures in a pregnant patient?

Concerns of laparoscopy during pregnancy:
- Risk of premature labor or miscarriage
- Risk of damaging gravid uterus.

**Recommendations for safe laparoscopic procedure in pregnant patients:**
- The operation should be done during second trimester, ideally before 23rd week of pregnancy to minimize the risk of miscarriage or preterm labor and maintain adequate intra-abdominal working room.
- Tocolytics are beneficial to arrest preterm labor.
- Uterine fundus reaches umbilicus at 20 weeks, which should be taken into consideration during abdominal access for creation of pneumoperitoneum. Direct peritoneal access (Hasson's technique) should be used to avoid damage to uterus or its blood supply.
- Pregnant patient should be positioned with left lateral tilt to avoid aortocaval syndrome.
- Pregnancy possesses risk of thromboembolism, so sequential compression devices are essential.
- Fetal monitoring should be performed using transvaginal ultrasound.
- Mechanical ventilation must be adjusted to maintain normal $EtCO_2$. Gasless laparoscopy is an alternative to avoid potential side effects of $CO_2$ pneumoperitoneum.

## Q. How Graft rejection is prevented?

Graft rejection is prevented by immunosuppression. Immunosuppressive drugs are used to diminish immunological attack on graft and target T cell activation. Immunosuppressive drugs used for prevention and treatment of acute and chronic rejection of transplanted organ therapy must be maintained lifelong.

Success of transplantation is largely due to effective immunosuppression. A typical regimen consists of different phases:
- *Induction:* Drugs administered immediately post-transplant to induce immunosupression. Biologic agents consisting of antibody preparations that are directed at various cells or receptors involved in rejection process are commonly used in induction.
- *Maintenance:* Drugs administered to maintain immunosuppression after recovery of the recipient from operative procedure. Nonbiological agents form the mainstay of maintenance protocol.

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Nonbiological agents are: Corticosteroids, azathioprine, cyclosporine, tacrolimus, sirolimus and mycophenolate mofelil.
Biological agents are: Polyclonal antibodies, monoclonal antibodies.

### Q. What are the categories of organ donors?

Two categories: (1) Deceased donors and (2) living donors

1. *Deceased donors:* These donors are deceased individuals meeting the criteria of brain death whose organs are being perfused by life support measures till the procurement of the organ.
2. *Living donors:* These donors are generally very healthy and almost exclusively classified as ASA 1 or 2 patients. The surgeon generally operates on the individual to procure the organ. These donors should undergo an extensive medical, psychological and social evaluation. For renal transplant, laparoscopic live donor nephrectomy has completely replaced the traditional open approach. A major concern about living organ donation is the potential for harm to the entirely healthy individual who undergoes major surgery for the interest of other.

### Q. Why and how organ preservation is done after procurement?

Organ harvesting and transplantation cause organ injury to the donor organ during three main periods:

1. Storage of recovered organ during transport
2. Implantation phase
3. Initial reperfusion phase with reintroduction of oxygen.

Hence, organ preservation is essential.

Most common method of preservation involves the use of hypothermia and pharmacological inhibition to slow down metabolic process in the organ after removal from the donor. Cold solutions have been developed to improve organ preservation by decreasing the detrimental effects of hypothermia alone. Most solutions are modified with additives (e.g. oxygen radical scavengers) that are believed to improve organ storage condition. Maximum cold preservation time varies among organs and solutions used. Kidneys can be stored in cold preservation solution for up to 24–30 hours before transplantation.

### Q. What is end stage renal disease? What is physiology of ESRD?

End stage renal disease (ESRD) is the term used to describe a clinical syndrome characterized by multiple organ dysfunction that will prove fatal without dialysis. Patients with chronic renal failure are at increased risk of developing ESRD. They have glomerular filtration rates of <25% of normal with an increased blood nitrogen waste (urea) or retention of fluid and electrolytes.

**Pathphysiology of ESRD:** In chronic renal failure, there is extreme decrease of functioning nephrons and GFR which results in inability of kidney to

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regulate the volume and composition of extracellular fluids and excrete waste products. The loss of homeostasis, accumulation of cellular toxins and inability of the kidney to maintain water balance causes multiple organ system dysfunction.

When urine production declines to less than 400 mL/day, patient becomes oliguric and fails to excrete dietary fluid and electrolyte load. Abnormalities in $Na^+$, $K^+$, $Ca^{++}$, $Mg^{++}$ and phosphate levels develop, with most threatening being hyperkalemia. These patients require frequent or continuous dialysis.

Metabolic acidosis occurs in two forms: Hyperchloremic normal anionic gap acidosis and high gap acidosis due to inability to excrete titrable acids by the kidney.

Cardiovascular complications of ESRD are due to volume overload, high renin angiotensin activity, autonomic nervous system dysfunction, acidosis and electrolyte disturbances. Hypertension is almost a universal finding in ESRD contributing to development of myocardial dysfunction and heart failure (chronically elevated systemic pressure loads to left ventricular hypertrophy and increased myocardial oxygen requirement). Pericarditis may occur secondary to uremia. Dilated cardiomyopathy and concentric hypertrophy can develop in response to increase in intravascular volume and afterload. The accumulation of uremic toxins and metabolic acidosis contribute to poor myocardial performance. ESRD with depressed ventricular function is not contraindication to renal transplantation but may complicate anesthetic management. Renal failure also accelerates progression of atherosclerosis in coronary arteries. Arrhythmias may be encountered in patients with ESRD due to electrolyte abnormalities.

Pulmonary edema and restrictive pulmonary dysfunction are common patients with renal failure which are responsive to dialysis. Hypervolemia, heart failure, reduced serum oncotic pressure and increased pulmonary capillary permeability are factors contributing to pulmonary edema.

Anemia of chronic renal failure is due to reduced level of erythropoietin, red cell damage, and ongoing gastrointestinal blood loss and iron or vitamin deficiencies. It is of normochromic, normocytic variety and treated with recombinant erythropoietin.

A qualitative defect in the platelet function reduced by uremia aggravates blood loss, treated by cryoprecipitate and desmopressin acetate.

A thromboelastographic study of whole blood clotting found increasing coagulability and decreased fibrinolysis resulted from uremia. Uremia causes CNS disturbances as drowsiness, memory loss, myoclonus seizures, stupor and coma. Signs of peripheral or autonomic neuropathy are strong indications for dialysis.

Chronic uremia causes decreased gastric emptying. All presenting for kidney transplantation should be considered as full stomach.

Acquired defects in both cellular and humoral immunity account for high incidence of serious infection and high mortality due to sepsis in CRF patients.

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## Q. What is renal failure? How is it categorized?

The term renal failure is used to denote failure of renal excretion leading to retention of nitrogenous waste products of metabolism including creatinine and urea. Other aspects of renal function including regulation of fluid and electrolyte status and endocrine function of kidney also fail.

The most fundamental categorization of renal failure is acute and chronic type.

## Q. What is acute renal failure?

Sudden inability of the kidney to vary the urine volume and content appropriately in response to homeostatic needs. There are three main types depending on etiology:

1. *Prerenal:* Results from decreased renal perfusion due to circulatory problems.
2. *Renal:* Results from primary or secondary renal disease, toxins and pigments.
3. *Post-renal:* Results from obstruction of urinary tract.

Pre-renal and Post-renal types are usually reversible with treatment. Renal type is most serious and often needs hemodialysis.

**Classification according to urine flow rate:** Oliguric, non-oliguric, polyuric renal failure.

In acute oliguric renal failure, there is progressive increase in creatinine and BUN concentration and urine flow <20 mL/hour.

## Q. What are the presenting features of acute renal failure?

- Patients are oliguric (urine <500 mL/day)
- Anorexia, nausea, vomiting
- Muscle twitching
- Drowsiness, confusion and coma
- Increased respiratory rate due to acidosis, pulmonary edema and respiratory infection
- Anemia due to blood loss resulted from platelet dysfunction and disturbance of coagulation
- Severe infection due to depressed cellular and humoral immune mechanism
- Increased plasma creatinine and urea concentration. Hyperkalemia with metabolic acidosis.

## Q. What is the management of acute renal failure?

- Hyperkalemia ($K^+$ >6 mmol/L) should be treated immediately
- Optimization of circulatory blood volume to be done for adequate renal perfusion using CVP and pulmonary wedge pressure (PWP) wedge monitoring
- Acidosis corrected with isotonic sodium bicarbonate
- Inotropic drug administration to restore effective blood pressure.

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**Treatment of hyperkalemia ($K^+$ >7 mmol/L):**

| Therapy | Mechanism |
|---|---|
| • Intravenous calcium gluconate (10 mL of 10% solution), if ECG changes suggest hyperkalemia | Stabilization of cell membrane potential |
| • Inhaled $\beta_2$ agonist e.g. salbutamol<br>• Intravenous glucose (50 mL of 5% solution) and insulin 5U<br>• Intravenous sodium bicarbonate (100 mL of 8.4% solution) if acidosis present | Shift $K^+$ into cells |
| • Intravenous furosemide and NS, if adequate residual renal function<br>• Ion exchange resin<br>• Dialysis | Remove $K^+$ from body |

Early treatment cures the condition without any residual renal impairment. With delayed and ineffective treatment, renal failure is established and renal replacement therapy (RRT) required.

## Q. What is chronic renal failure? What are the causes?

Chronic renal failure (CRF) is an irreversible deterioration of renal function which develops over a period of years. Initial manifestation is biochemical abnormality. Eventually loss of secretory, metabolic and endocrine functions of kidneys result, producing signs and symptoms of CRF (uremia).

When death is likely without renal replacement therapy, it is called end stage renal failure.

**Causes:** CRF is caused by any condition, which destroys the normal structure and functions of the kidney.

- Congenital (polycystic kidney)
- Renal artery stenosis
- Hypertension
- Glomerular diseases (IgA nephropathy is the most common)
- Diabetes mellitus.

## Q. What investigations are done in a patient with CRF?

- Full blood count (anemia)
- Blood urea, creatinine and electrolyte concentrations
- Blood calcium, phosphate and albumin concentrations
- Blood lipid and glucose
- Renal ultrasound
- Chest X-ray (heart size and pulmonary edema)
- ECG in patients above 40 years and with risk factors for cardiac disease
- Renal artery imaging
- Hepatitis and HIV serology.

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## Q. What are the considerations for the management of CRF?

- Identification of underlying renal disease and their treatment
- Prevention of further renal damage by:
  - Control of systemic blood pressure to retard deterioration of CRF. The target BP is 130/85 mm Hg. ACE inhibitors are more effective in retarding the progress of renal failure
  - Moderate restriction of dietary protein to 60 g/day is recommended. This should be accompanied by adequate calorie intake to prevent malnutrition.

  Once the plasma creatinine exceeds 3.4 mg/dL, there is progressive deterioration of renal function irrespective of etiology
- Prevention of adverse effects due to loss of renal function
- Institution of renal replacement therapy (dialysis, transplantation) when appropriate.

## Q. What are the adverse effects of CRF? How to limit them?

- *Anemia:* Correlates with the severity of renal failure and contributes to many of the nonspecific symptoms of CRF. Recombinant human erythropoietin is effective in curing anemia of CRF.
- *Fluid electrolyte imbalance:* Failing kidney is unable to concentrate urine, a relatively high volume is needed to excrete products of metabolism. Hence, a fluid intake of 3 L/day is desirable. Some patients with salt wasting disease require an intake of 5–10 g/day of sodium chloride.
- *Acidosis:* Declining renal function is associated with metabolic acidosis. Acidosis causes further reduction in renal function and increased tissue catabolism. Plasma bicarbonate should be maintained above 22 mmol/L.
- *Cardiovascular disease and lipids:* Atherosclerosis is common and accelerated by hypertension. Pericarditis is common in ESRF. Hypertension and increased triglyceride level are common in patients with CRF, should be reduced by appropriate medications.
- *Infection:* Cellular and humoral immunity are impaired with increased susceptibility to infection with CRF, needs prompt treatment.
- *Bleeding:* Patients with CRF have increased bleeding tendency, manifested as ecchymoses. Adequate dialysis corrects it partially.
- *Renal osteodystrophy:* It is metabolic bone disease in CRF that consists of osteomalacia, osteosclerosis, and hyperparathyroid bone disease. To minimize the effect of CRF on bone, plasma calcium and phosphate levels are kept nearer to normal values.
- *Myopathy:* Generalized myopathy is combined effect of poor nutrition, hyperparathyroidism, vitamin D deficiency and disorders of electrolyte metabolism. Proper correction is needed.
- *Other side-effects:* Neuropathy is due to demyelination of medulated fibers. Motor neuropathy—foot drop, autonomic neuropathy—delayed gastric emptying and postural hypotension.

  Neuropathy is a late manifestation of CRF and improves or resolves with dialysis.

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Half-life of insulin is prolonged in end stage CRF due to decreased tubular metabolism and so insulin requirement declines in these patients. There is also insulin resistance.

GI manifestations are anorexia, nausea and vomiting.

### Q. What is renal repacement therapy?

Routine treatment of ARF and CRF to replace some functions of the kidney artificially by dialysis. It does not replace endocrine and metabolic functions of the kidney but aims to maintain plasma biochemistry (uremic toxins, electrolytes and acid-base status) at acceptable level. Dialysis also removes fluid from circulation. Hemodialysis is common form of RRT.

### *Q.* What are the common types of RRT in CRF?

**Intermittent hemodialysis:** Commonly used and standard blood purification therapy in ESRF. It is started when the patient has symptomatic advanced renal failure but before development of complications with plasma creatinine of 6.8–9 mg/dL. Vascular access is required, an arteriovenous fistula is formed usually in forearm or a prosthetic arteriovenous graft is inserted. Dialysis is carried out for 3–5 hours, three times weekly. The dialysis principle involves equilibriation of waste products in the patient's blood across a semipermeable membrane to the dialysis bath.

Both the patient and circuit are heparinized to prevent clotting. The signs and symptoms of uremia (volume overload, acid-base and electrolyte imbalance, abnormal mental function, muscle weakness, peripheral neuropathy and defective coagulation) are improved with this treatment. Hypertension is also improved.

If fluid and electrolyte shifts are too rapid, dialysis disequilibrium occurs with manifestations of nausea, vomiting, weakness, convulsion and coma.

Vascular access is done under local filtration, brachial plexus block (axillary) or general anesthetic uremic patients are debilated and should be given smaller doses of all drugs including local anesthetics. Brachial plexus block produces analgesia and vasodilatation facilitating insertion of cannula. Duration of block is shorter in uremic patients. Epinephrine content of local anesthetic should not be more than 1:200,000 and dilution of 1:400,000.

**Problems during hemodialysis:**

- *Hypotension:* Sudden decrease of BP due to fluid removal and hypovolemia. Treated by saline infusion.
- *Cardiac arrhythmias:* Decreased BP, chest pain due to acid-base balance shift. Dialysis is stopped and correction done.
- *Hemorrhage:* Due to anticoagulation. Dialysis is stopped and treatment instituted.
- *Dialysis hypersensitivity:* Due to allergic reaction to dialysis membrane, presenting with acute circulatory collapse. Dialysis is stopped.
  Emergencies between treatments:
  - Pulmonary edema (breathlessness)
  - Systemic sepsis (rigor, fever and decreased BP).

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**Continuous ambulatory peritoneal dialysis (CAPD):** Patient's peritoneum acts as exchange membrane and vascular access is not needed. It is less efficient and dialysis time is longer. The dialysis involves insertion of a permanent silastic catheter into peritoneal cavity. Isotonic dialysis fluid is introduced and left in place for 6 hours and then drained. The cycle is repeated 4 times daily. Patient remains ambulatory. Concerns are pain during dialysis and chance of peritonitis. Automated peritoneal dialysis is now widespread, where the fluid exchange is performed at night with a mechanical device leaving the patient free or with a single exchange during the day.

## Q. What are the indications of RRT in ARF?

Indications are:

- *Increased plasma urea and creatinine:* Urea >180 mg/dL, creatinine >6.8 mg/dL and progressive biochemical deterioration with little or no urine output.
- *Hyperkalemia:* Potassium level >6 mmol/L is hazardous and is usually reduced by medical treatment but dialysis is often required for definite control.
- *Metabolic acidosis:* Further raises plasma $K^+$ level
- *Fluid overload and pulmonary edema:* With presence of urine output, it is controlled by careful fluid balance and use of diuretics. In oliguria or anuria, RRT is indicated.
- Uremic pericarditis and uremic encephalopathy occur in severe, untreated RF and are strong indications for RRT, uncommon in ARF.
- *Options of RRT in acute renal failure:* Hemodialysis, high volume hemofiltration, continuous arteriovenous or venovenous hemofiltration and peritoneal dialysis.

## Q. What is the indication of kidney transplantation? How is it performed and preserved?

Kidney transplantation is indicated in and offers best chance of long-term survival for patients with ESRD. It restores normal kidney function and corrects all metabolic abnormalities of CRF.

Renal transplantation with organs from living donors is increasing rapidly in recent years. The donor kidney is usually implanted in the iliac fossa with no need for native nephrectomy. The donor vessels are anastomosed to the recipient iliac artery and vein and the donor ureter to the bladder. ABO blood group compatibility between donor and recipient is essential. A cytotoxicity test for antibodies against HLA antigens and T and B cells, cross-matching tests are performed pretransplant. After transplant, immunosuppressive therapy is required to prevent graft rejection and is to be continued throughout life. Triple therapy with prednisolone, cyclosporin/tacrolimus and azathioprine is commonly used. Prolonged use of immunosuppression is associated with increased incidence of infection and increased risk of malignancy. Newer drugs such as mycophenolate mofetil, rapamycin are increasing in use. The goal of therapy of the newer drugs includes:

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- Prevention of immune response
- Prevention of complications of immunosuppression as opportunistic infection and malignancy
- Minimization of drug-induced toxicity.

## Q. What are the complications of chronic immune suppression?

| Systems | Complications |
|---|---|
| Central nervous system | Lowered seizure threshold |
| Cardiovascular system | Hypertension, hyperlipidemia, diabetes |
| Renal/electrolyte | Decreased GFR, hyperkalemia, hypomagnesemia |
| Hematologic/immune | Increased risk of infection, increased risk of tumor, pancytopenia |
| Endocrine/others | Osteoporosis, poor wound healing |

## Q. What are the functions of the kidney?

- Regulation of volume and composition of body fluids by making large volume of ultrafiltrate of plasma (120 mL/min, 170 L/day) at glomerulus and selectively absorbing components from it at points along the nephron.
- Excretion of metabolic breakdown products (urea, creatinine and ammonia), drugs and toxins.
- Regulation of acid-base balance.
- Secretes rennin (from juxtaglomerular apparatus), which generates angiotensin II.
- The kidney is main source of erythropoietin.
- The kidney is essential for vitamin D metabolism.

## Q. What are the tests for assessment of kidney function?

- *Urine specific gravity:* As renal function decreases, the ability of the kidney to concentrate urine decreases and is reflected by proportional change of specific gravity (normal: 1.012).
- *Serum creatinine level:* It is the end product of muscle creatine metabolism and excreted by kidney. It does not reflect early loss of renal function, because serum level remains normal until 50% loss of kidney function (Normal: 0.8 mg/dL).
- *Endogenous creatinine clearance rate:* It is the best measure of kidney function and is defined as the volume of plasma from which creatinine is completely removed per unit of time. (Normal: 90 to 110 mL/min). It provides a simple, inexpensive, bedside estimate of CRF. Small amount of creatinine is secreted by proximal tubules.
- *Inulin clearance:* Inulin is an inert polyfructose sugar that is completely filtered by the glomerulus and is neither secreted nor absorbed by renal tubules. Therefore, inulin clearance is the gold standard for measuring GFR, yet it is seldom used clinically as its accurate measurement is laborious and requires meticulous attention to detail.
  Normal values in males: 110 to 140 mL/min; in females: 95 to 125 mL/min.

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Renal function is assessed preoperatively with serum electrolytes, blood urea nitrogen and creatinine measurements. Information about the size and perfusion are obtained by CT scan or aortogram.

Perioperative medication and perfusion strategies are adjusted based on renal function.

### Q. What are the considerations in preoperative evaluation of a kidney transplanted patient?

When renal transplanted patients have subsequent surgery, the status of their renal function must be determined. Good graft function is determined by BUN and creatinine values and sufficient urine volume. Renal function must be assessed, as long-term immunosuppression leads to chronic renal insufficiency. The presence of graft rejection should be evaluated.

These patients are still at higher cardiac risk due to persistent risk factors as diabetes, hypertension, dyslipidemia and secondary hyperparathyrodism. Congestive heart failure (cardiomyopathy), left ventricular hypertrophy and ischemic heart disease remain important complications in renal transplanted patients. Therefore, a careful cardiac evaluation is essential.

Immunosuppressant therapy results in hyperglycemia, adrenal suppression (steroids), increased risk of infection, hypertension, renal insufficiency (cyclosporine, steroids and tacrolimus), myelosuppression with anemia, thrombocytopenia, and leukopenia (azathioprine, sirolimus). The side effects of immunosuppressants should be sought and close monitoring of blood glucose level and cardiovascular function is done.

Dosage scheduled for all medications should be noted and the patient is instructed to continue all the drugs.

### Q. What are the considerations during anesthetic management of a kidney transplanted patient?

Both regional and general anesthesia are used successfully. Nasal intubation is avoided for risk of infection by nasal flora. Standard ASA monitors are used. Invasive monitoring not indicated solely because of previous transplantation and usually avoided due to increased risk of infection resulted from immunosuppression therapy.

A major consideration for renal transplanted recipient is maintenance of renal perfusion with adequate volume replacement. The CVP monitoring during volume replacement is useful for preventing prerenal damage to transplanted kidneys and the line should be placed using strict aseptic technique.

Antibiotics, antiviral, antifungal and immune suppression regimens should be disrupted as little as possible in the perioperative period.

The specific issue in renal transplanted patients is that, despite presence of normal creatinine level, GFR is generally decreased leading to electrolyte abnormalities and altered drug metabolism.

Nephrotoxic drugs, NSAIDs and COX-2 inhibitors should be avoided in renal transplanted patients.

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Drug interaction between immunosuppressants and anesthetic drugs may occur. Cyclosporine alters barbiturate, fentanyl, and isoflurane requirement. It also enhances the effect of neuromuscular blocking (NMB) drugs leading to decreased requirement of non depolarizing muscle relaxants in patients taking cyclosporine.

Azathioprine causes small and transient antagonism to nondepolarizing muscle relaxants. Produces no effect on atracurium requirement.

Preoperative review of patient's medication focuses on possible side effects.

DVT prophylaxis should be considered in renal transplanted patients. As there is increased risk of infection, patient cross-contamination should be avoided.

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# Case Discussion on Anesthesiology

*Salient Features*

- Written especially for the postgraduate students of DND and MD Anesthesiology
- Purpose of this book is to help the postgraduate students to be familiar with the questions asked during viva on long and short cases
- Contains discussion on ten long cases and fourteen short cases, commonly presented during practical examination
- Presents case discussion in question-answer format in a sequential manner
- Aimed at providing the manner of answering during case presentation in viva.



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