



# Human Leukocyte Antigens Sensitization in Kidney Transplant: Its Prevention and Challenges

## Abstract

The presence of donor-specific antibodies (DSAs) against human leukocyte antigens (HLA) indicates sensitization and poses a major barrier to successful transplantation. Sensitization may result from pregnancy, blood transfusion, prior transplantation, or infection, making it difficult to identify compatible donors and often prolonging waiting times. DSAs may be performed or developed *de novo* after transplantation. Preformed DSAs, generated from previous HLA exposure, are associated with early antibody-mediated rejection (ABMR). In contrast, *de novo* DSAs arise post-transplant due to HLA mismatches, inadequate immunosuppression, nonadherence, or graft inflammation, and are a key cause of late rejection and chronic graft injury. Advances in detection techniques, particularly solid-phase assays such as ELISA and Luminex single-antigen bead testing, have improved sensitivity and risk assessment. Preventive strategies include minimizing blood transfusions, using leukoreduced or HLA-matched blood products, and applying refined matching approaches such as eplet analysis to reduce alloimmunization and improve long-term transplant outcomes.

**Keywords:** Blood transfusion, Donor-specific antibody, HLA sensitization, Pregnancy, Renal transplantation

## Introduction

The presence of antibodies directed at donor human leukocyte antigens (HLA) indicates sensitization. These donor-specific antibodies (DSAs) have the potential to trigger acute graft rejection and chronic graft nephropathy.<sup>1,2</sup> DSAs can be identified before kidney transplant (preformed DSA) or be identified after transplant (*de novo* DSA). Preformed DSAs are formed after previous exposure to HLA, generally through pregnancy, wherein paternally inherited fetal antigens are exposed to the mother's blood, blood product transfusion, previous transplantation, or remotely due to previous infection and vaccination. These antibodies are commonly directed against HLA class I molecules (HLA-A, HLA-B, HLA-C). These are expressed on all nucleated cells, are of IgG1 and IgG3 subtypes, and have strong complement binding capacity. These antibodies can increase the risk of rejection early in the course of transplant, manifesting as hyperacute rejection, accelerated acute rejection, or active antibody-mediated rejection (ABMR).<sup>3</sup>

*De novo* DSAs can be formed due to risk factors like high HLA mismatches,

especially DQ mismatches, nonadherence or inadequate immunosuppression, and graft inflammation due to viral infection or ischemia-reperfusion injury, which in turn leads to increased graft immunogenicity. These antibodies are predominantly directed against HLA class II molecules (HLA-DP, HLA-DQ, HLA-DR), present on antigen-presenting cells (APCs) like macrophages, dendritic cells, B cells, and also endothelial cells after inflammatory insult. They appear late in the course of transplant, are of IgG2 and IgG4 subtypes, and have weak or no complement-binding capacity. They cause late rejection like late ABMR, chronic ABMR, and transplant glomerulopathy.<sup>4</sup>

## Methods of Detection of DSAs

The detection of DSAs has evolved considerably over time, progressing from traditional cell-based assays to highly sensitive solid-phase technologies. Historically, complement-dependent cytotoxicity crossmatch (CDC-XM) was the primary method. In this assay, recipient serum is incubated with donor T and B lymphocytes, followed by the addition of complement. If complement-fixing antibodies are present, they trigger cell

**Abhilasha Soni<sup>1</sup>, Rajesh Jhorawat<sup>1</sup>, Manish Chaturvedy<sup>1</sup>**

<sup>1</sup>Department of Nephrology, AIIMS Jodhpur, Jodhpur, Rajasthan, India

**Corresponding author:** Rajesh Jhorawat, Department of Nephrology, AIIMS Jodhpur, Jodhpur, Rajasthan, India.  
E-mail: jhorawat2000@gmail.com

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lysis, which is visualized under ultraviolet microscopy. CDC-XM can detect both HLA and non-HLA antibodies of IgG and IgM classes; however, it primarily identifies complement-binding antibodies. Although CDC-XM has a high positive predictive value for predicting early ABMR, its overall sensitivity is low.<sup>5</sup>

Flow cytometry crossmatch (FC-XM) was introduced subsequently. FC-XM detects IgG antibodies directed against HLA and non-HLA antigens with greater precision than CDC-XM (5). However, it does not differentiate between complement-fixing and non-complement-fixing antibodies, limiting its ability to predict complement-mediated injury. Currently, solid-phase assays are considered the gold standard for HLA antibody screening. These include ELISA-based methods or Luminex single-antigen bead assays. Luminex technology offers superior sensitivity and specificity and enables identification of antibodies against specific HLA loci, particularly HLA-DQ, -DP, -DR, and -Cw. Antibody strength is measured semi-quantitatively using mean fluorescence intensity (MFI).<sup>6</sup> Certain therapies containing immunoglobulins, such as intravenous immunoglobulin (IVIG), antithymocyte globulin (ATG) and rituximab, may interfere with assay accuracy and yield falsely low MFI readings.<sup>6,2</sup> Studies have shown higher DSA positivity and antibody strength following previous transplantation compared with pregnancy or blood transfusion.<sup>7</sup> Luminex testing remains limited to specialized centers due to high costs, limited technical expertise, and logistical challenges.

## Causes of HLA sensitization

### A. Pregnancy

Pregnancy is the most common natural cause of alloimmune sensitization. The clinical relevance of pregnancy-related sensitization was first identified in the 1950s by Van Rood and colleagues during investigations of transfusion reactions in peripartum women.<sup>8</sup> Exposure to fetal antigens inherited from the father can stimulate the maternal immune system, leading to activation of B lymphocytes and the production of alloantibodies against these non-self HLA antigens. Population-based studies estimate that ~24-49% of women with past pregnancy develop detectable alloantibodies.<sup>7</sup> The number of pregnancies, the interval between delivery and antibody testing, and the sensitivity of the detection method used influence the prevalence and magnitude of sensitization.<sup>7,8</sup>

Anti-HLA antibodies are detected in about 30% of pregnant women using CDC assays and up to 50-70% using more sensitive single-antigen bead assays. Antibody levels peak within the first 90 days postpartum and decline over the following 1-2 years.<sup>9</sup> Screening studies among female blood donors have demonstrated anti-HLA antibodies in 17.3% of all female donors and 24.4% of those with prior pregnancies, with prevalence increasing with higher parity. In contrast, sensitization rates among men remain low and show minimal association with transfusion history.<sup>10</sup>

A recent study found that pregnancy-induced antibodies were present in 75% of women awaiting kidney transplantation, with significant reactivity against paternal HLA epitopes.<sup>11</sup> Despite maternal immune tolerance toward the fetus, immune activation may still occur through indirect antigen presentation in lymphoid tissues, a phenomenon known as the immunological paradox of pregnancy.<sup>9</sup> A study from India reported anti-HLA antibody detection rates of 57.46%, 70.14%, and 18.85% using Luminex-panel reactive antibody (PRA), FC-XM, and CDC-XM, respectively.<sup>7</sup>

### B. Infection and immunization

Patients with CKD have an increased risk of infections due to uremia-associated immune dysfunction, catheter-related bloodstream infections, invasive procedures, and repeated hospitalizations. Infections may contribute to alloimmunization, complicating future transplantation.<sup>12</sup> Red cell alloimmunization was associated with tissue-invasive bacterial infections, prolonged fever, and disseminated viral infections, but not with uncomplicated infections, gram-positive bacteremia, fungal infections, elevated C-reactive protein, or leucocytosis.<sup>13</sup> Vaccination may rarely trigger alloimmune responses. Proposed mechanisms include activation of T- and B-cells, cytokine-mediated stimulation of dormant alloreactive memory cells, and nonspecific immune activation by adjuvants. However, a meta-analysis by Mulley showed low rates of DSA formation (1.85%) and rejection (2.1%), confirming that vaccination benefits outweigh potential risks.<sup>14</sup>

### C. Blood transfusion

Blood products, especially leukocytes, contain HLA antigens. Lymphocytes constitute a significant portion of the antigenic load in a blood unit, whereas monocytes, in addition to class-I, also express class-II HLA antigens. Individuals positive for HLA-B7 or on interferon-alpha therapy exhibit higher HLA expression. Platelets also express HLA molecules.<sup>15</sup>

A large meta-analysis by Hassan examined data from over 32,000 kidney transplant recipients. The median transfusion prevalence was 40% (range 18-64%). Transfusions were linked to worse outcomes, including lower survival rates, increased organ loss, rejection episodes, and the development of DSAs.<sup>16</sup> Among the 10 included studies, unadjusted data on allograft survival were available in seven, encompassing 28,673 patients. These studies revealed that patients who received post-transplant blood transfusions had a higher likelihood of allograft failure than those who did not, with an odds ratio (OR) of 2.11 (95% CI: 1.69-2.64). Additionally, 9/10 studies involving 20,258 patients showed that patients who received a transfusion had 1.42 times the chance of rejection compared to those who did not. Furthermore, 8/10 studies, representing 19,000 patients, indicated that post-transplant blood transfusion was associated with

higher odds of DSA detection than no transfusion, with an OR of 1.73 (95% CI: 1.24-2.41)<sup>16</sup>.

#### D. Graft Loss

Many patients who lose their graft develop HLA antibodies, making it harder to find a compatible kidney for a future transplant.<sup>2</sup> Even if an individual tests negative for HLA antibodies at the time of graft loss, they can later become positive due to factors such as discontinuation of immunosuppressants or exposure to pro-inflammatory events like blood transfusions or infection.<sup>2</sup> For organ transplantation other than kidneys (e.g., heart), where size and minimizing tissue damage (ischemia-reperfusion injury) are critical factors, HLA matching may not be the top priority during transplantation. This less stringent approach can lead to a high prevalence of HLA antibodies in recipients, ranging from 12% to 92%.<sup>2</sup> Re-transplantation is strongly associated with both class-I and class-II HLA antibodies.<sup>7</sup> US Renal Data System (USRDS) reports that around 4-5% of the incident dialysis population has a failed kidney allograft and represents an important portion of people wait-listed for kidney transplantation (15%).<sup>17</sup> A similar trend was also described in the national French Renal Epidemiology and Information Network (REIN) registry.<sup>18</sup>

#### Prevention and challenges

##### A. Preventing sensitization from pregnancy

Triulzi *et al.* reported HLA antibodies in 17.3% of all female donors (n = 5834) and in 24.4% of those with a history of pregnancy (n = 3992).<sup>10</sup> Even abortions or miscarriages increase HLA sensitization, and the risk is proportional to their number.<sup>19</sup> Proper counseling of patients with CKD on contraceptive measures is very important to prevent HLA sensitization. The following strategies can be employed to avoid or decrease HLA sensitization: avoiding multiple pregnancies, avoiding blood transfusions during pregnancy, avoiding pregnancy in patients with CKD using proper contraceptive measures, and providing vigilant prenatal care with prompt treatment of conditions such as miscarriages or abortions that could trigger immune responses and HLA sensitization. HLA sensitization in female patients with CKD affects subsequent renal transplant outcomes, even when they undergo a perioperative desensitization procedure, compared with their male counterparts.<sup>20</sup>

Fertility after successful renal transplantation improves dramatically compared to women with CKD. No increased frequency of *de novo* DSAs after delivery in renal transplant recipients was detected in the pregnancy group (n = 40) compared to the control (n = 40) in the study reported by Kaatz *et al.*<sup>21</sup> Overall, pregnancies in women after kidney transplantation showed good allograft and maternal outcomes in this study. Even in the meta-analysis by Buren *et al.*, it was shown that pregnancy after KT does not affect long-term graft survival compared to nulliparous controls. Systematic review of the literature showed that mainly pre-pregnancy proteinuria, hypertension, and high

serum creatinine are risk factors for graft loss rather than pregnancy itself.<sup>22</sup> The safe time for pregnancy after renal transplantation is considered after 1 year; however, a meta-analysis by Shah *et al.* has shown that neonatal outcomes are better after 3 years post-renal transplantation.<sup>23</sup> Therefore, preconception counselling, family planning, and contraception are pertinent parts of CKD and in the renal transplant counselling process.

##### B. Preventing sensitization from transfusion

Several methods can help prevent HLA alloimmunization in blood transfusions. These include leukoreduction (LR), irradiation, pathogen reduction technology (PRT), or HLA-matched blood. LR reduces the number of white blood cells (WBCs). Irradiation (UV-B irradiation) seems to deactivate Class II molecules found on donor APCs. Varied wavelengths of illumination are employed alongside a photochemical agent for damaging the pathogen's DNA in blood in PRT. To avoid the need for blood transfusion, the concept of patient blood management (PBM) was developed. The three pillars of PBM are as follows: (i) detection and management of anemia, (ii) minimization of blood loss and bleeding, and (iii) leveraging and optimizing the patient-specific physiological tolerance of anemia [Table 1].<sup>24</sup>

Despite PBM, HLA sensitization following blood transfusion requires attention. LR in the number of leukocytes, in allogenic blood products, has been proven to be clinically useful.<sup>25</sup> The accepted standard of leukocyte-depleted blood components has been mentioned in Table 2.

New techniques, such as Eplet matching or Luminex, are being used in high-resource settings. However, in resource-limited conditions, selective pre-storage leukofiltration for patients on regular transfusions will achieve 3-4 log leukoreduction. The approach to reduce leukocytes and to decrease allosensitization through blood products can be stratified.

- High-resource setting: Luminex, eplet matching, universal leukoreduction
- Moderate-resource: Selective leukoreduction, CDC/flow crossmatch
- Low-resource: PBM protocols, restrictive transfusion thresholds, referral for sensitized patients

Harvesting blood components through apheresis technology typically results in 3-4 log leukoreduction and provides better therapeutic benefits than random donor products.<sup>25</sup>

##### C. Preventing sensitization from transplantation

Transplantation is a significant risk factor for HLA sensitization, affecting both class I and class II DSAs. Infections, rejection, and non-compliance with immunosuppression are important contributors to HLA sensitization.<sup>16</sup> In addition, tapering of immunosuppressants or allograft nephrectomy in a failing graft has been associated with the risk of HLA sensitization. More

**Table 1: Components of patient blood management**

1. Optimize erythropoiesis
Pre-transplant:
Optimize anemia management
Peri-transplant:
EPO use
Iron use
Medication review
Treat infections promptly
Timely arteriovenous fistula creation to decrease the risk of Infection
2. Minimize blood loss and bleeding
Intraoperatively:
Optimize coagulation parameters
Cell salvage (high risk of bleeding)
Peri-transplant:
Avoid over-anticoagulation
Judicious phlebotomy
3. Optimize the patient's physiological reserve in relation to anemia (avoid unnecessary transfusions)
Pre-transplant:
Optimize medically
Peri-transplant:
Optimize fluid status
Restrictive transfusion thresholds
Single units of blood were required

and Fusion MatchMaker, can produce different eplet mismatch loads and results for the same donor-recipient pair. Eplet matching requires high-resolution HLA typing, which may not be feasible in resource-limited conditions and deceased donor renal transplantation. Despite these limitations, new evidence is evolving using a combination of different molecular mismatch assessment approaches, such as eplet mismatch count, the number of highly immunogenic eplets, and PIRCHE-II score, to overcome specific technique limitations.<sup>53</sup>

### **ii. Role of weaning from immunosuppression after graft failure**

Second renal transplantation, both live or deceased, has shown better outcomes compared to continuing on dialysis.<sup>54</sup> However, the availability of repeat pre-emptive live or deceased donors is often limited; the continuation of immunosuppression with a failing graft raises concern, as it has a significant impact on preventing HLA sensitization and graft intolerance syndrome (GIS). However, on the other side, it increases the risk of infection and malignancy in dialysis patients. In a study by Augustine *et al.*, the proportion of highly sensitized patients increased from

**Table 2: Accepted standard of leukocyte-depleted blood components**

Agencies	Blood component (WB, PRBCs)	RDP for pooling
American Association of Blood Banks (USA)	WB, PRBCs, and Apheresis platelets	8.3 x10 <sup>5</sup> WBC/Unit <5x10 <sup>6</sup> WBC/Unit (red cell loss not more than 15%)
European Council criteria	<1x10 <sup>6</sup> WBC/Unit (Hb 40/unit)	≤2.0 x 10 <sup>5</sup> WBC/Unit
Director General of Health Services (India)	<5x10 <sup>6</sup> WBC/unit (red cell loss not more than 10%)	<8.3x 10 <sup>5</sup> WBC/Unit

RDP: Remote desktop protocol, PRBCs: Packed red blood cells, WB: Whole blood, WBC: White blood cells

advanced steps, like eplet matching, have the potential to decrease HLA sensitization further.<sup>11</sup>

### **i. The HLA matchmaker or Eplet matching**

Eplet matching is a novel approach revolutionizing the assessment of transplantation compatibility. Unlike traditional HLA matching methods that focus on antigen-level matching, the HLA matchmaker focuses on the polymorphic amino acid 4D configuration within the epitope, referred to as an Eplet, within the HLA molecules. It is particularly valuable in highly sensitized patients who have received multiple transfusions or undergone previous transplants, where conventional HLA matching methods may fall short.<sup>51,52</sup> Eplet matching offers a more accurate prediction of immune response, leading to reduced alloantibody formation and better graft survival rates. With this technique, transplant centers can select donors with the most compatible HLA profiles, increasing the likelihood of successful transplantation.<sup>51</sup> The main limitation of eplet matching is that not all eplets are equally immunogenic, which means high eplet mismatch does not always equate to a high immune response or rejection. The current eplet catalog is theoretically defined and not based entirely on proven immunogenicity. Different software and algorithms used for eplet analysis, such as HLA Matchmaker

21-68% after discontinuation of immunosuppression in a failing graft. Additionally, 41% of patients in the failing graft group required nephrectomy in the discontinuation group, whereas none in the immunosuppression group.<sup>55</sup>

Whether the patient is a candidate for re-transplantation determines how to wean off immunosuppression in a failing graft. Antimetabolites are first to be withdrawn at the initiation of dialysis, and subsequently, the CNIs are tapered to 50% at 3-6 months. The level of sensitization should be monitored with calculated PRA (cPRA) and symptoms of GIS. CNI can be stopped at 9-12 months. If there is no increase in cPRA, steroid can be withdrawn at 12 months or later. cPRA should be evaluated every 3-6 months, and withdrawal of all immunosuppression can be achieved if cPRA remains stable and signs and symptoms of GIS are absent.<sup>56</sup>

### **iii. Nephrectomy after graft failure**

The decision to perform a graft nephrectomy varies among centers. Most of the evidence is from retrospective studies, and there are center-to-center variations. GN has benefits like the ability to wean the patient off immunosuppression, remove source of chronic inflammation and creating

space for the third or fourth transplant, however, it is also associated with disadvantages like the loss of the residual functions of the failed graft including urine production, morbidity and mortality associated with the transplant nephrectomy especially in late and risk of HLA sensitization. The timing of allograft failure is important in making this decision [Figure 1].<sup>57,58</sup> Nephrectomy of early failed grafts (<6-12 months post-transplant) is associated with reduced HLA sensitization and improved subsequent transplant outcomes.

On the other hand, the removal of late failed grafts may increase detectable HLA antibodies, possibly because the failed transplanted kidney serves as a reservoir for *de novo* DSAs. While nephrectomy poses risks such as bleeding and sepsis, the preservation of residual renal function is associated with better cardiovascular outcomes. Also, if a failed allograft is retained, it can lead to a chronic inflammatory state or, in severe conditions, GIS, which is associated with anemia, erythropoietin resistance, and hypoalbuminemia.<sup>59</sup> Hence, graft nephrectomy should only be performed when there is a good indication, and non-elective surgery should be avoided, when possible, as it increases morbidity.

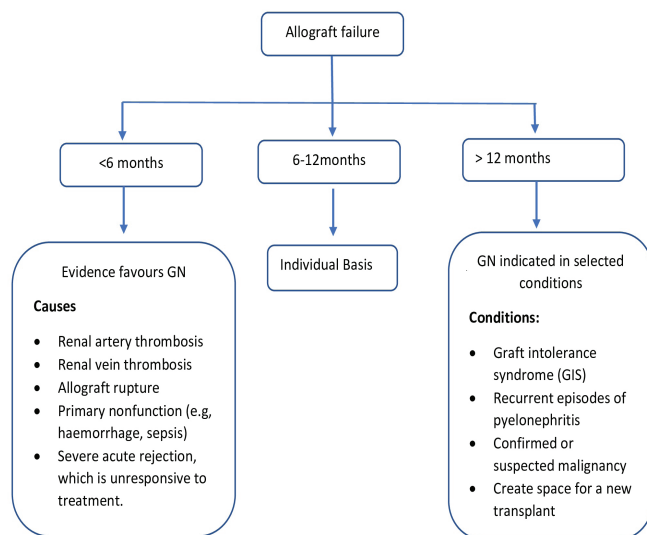


Figure 1: Approach to failed graft for graft nephrectomy (GN).

### Approach to a Sensitized Patient Awaiting Renal Transplantation

The primary concern at the time of transplantation is determining whether DSAs are present, as these antibodies may lead to hyperacute, accelerated, acute, or chronic graft rejection<sup>510</sup>. Transplant candidates can be categorized into different immunological risk groups, ranging from immunologically naive individuals to highly sensitized patients with cellular or serological memory [Table 3].<sup>511</sup> A consensus framework proposed by Furian and colleagues classified sensitization into distinct levels, enabling clinicians to tailor management strategies.<sup>511</sup>

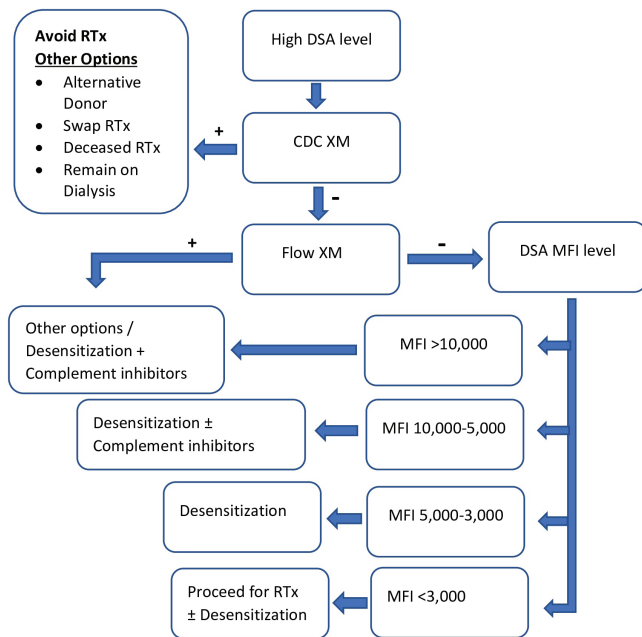
Assessing sensitization requires HLA typing and antibody screening using PRA and cPRA.<sup>512,513</sup> PRA measures the proportion of donor cells from a representative population that reacts with recipient antibodies, reflecting the degree of sensitization. In contrast, cPRA estimates the percentage of donors expected to be incompatible based on the recipient’s unacceptable antigens and their frequency in the donor population, as defined by allocation systems such as the Organ Procurement and Transplantation Network. CDC is used as a method to estimate PRA, whereas the Luminex method is used to calculate cPRA. A cPRA value greater than 80% indicates high sensitization and significantly reduces the chances of finding a compatible donor.<sup>511-513</sup> These methods are used to assess the level of sensitization in patients listed for deceased renal transplantation.

Since HLA antibody levels may fluctuate over time or increase following sensitizing events such as blood transfusion, pregnancy, or transplantation, periodic monitoring every 6-12 months is recommended.<sup>510</sup> Highly sensitized patients may require desensitization therapies, including plasma exchange, intravenous immunoglobulin, rituximab, and T-cell-depleting agents.<sup>511,513,514</sup> Emerging therapies, such as complement inhibitors and Imlifidase, an IgG-degrading enzyme, offer promising options for selected patients with persistent DSAs, improving transplantation opportunities and clinical outcomes.<sup>515</sup> Figure 2 highlights the approach in a patient with a high DSA level by SAB.<sup>511,516,517</sup>

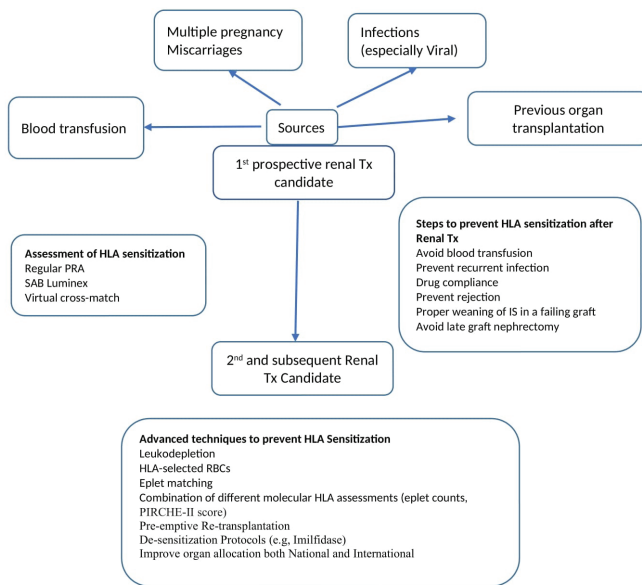
Table 3: Stratification for risk in kidney transplant recipients based on level of sensitization

Category	CDC	Flow	DSA	Memory type	Immunological risk	Renal transplant
1	+	+	+	Serological memory	Very high	Avoid
2	-	+	+	Serological memory	High	Better avoid/desensitization at the experience centre/Swap
3	-	-	+	Serological memory	High	Desensitization/Swap/Surveillance
4a	-	-	-	Cellular memory	Moderate (Previous Pregnancy/T ransplantation)	Surveillance with DSA, protocol biopsy
4b	-	-	-	Cellular memory	Mild to moderate (Previous blood transfusion)	Surveillance with DSA
5	-	-	-	Naive	Usual (No previous transplantation/pregnancy/ transfusion)	Usual risk

CDC: Complement-dependent cytotoxicity, DSA: Dono specific antibodies



**Figure 2:** Approach to a prospective renal transplant candidate patient with high DSA level by SAB. SAB: Single antigen bead, DSA: Donor-specific antibodies, RTx: Renal transplantation, MFI: Mean fluorescence intensity, XM: Crossmatch. (Note: DSA MFI level varies from laboratory to laboratory, transplant center should plan desensitization protocol as per their laboratory values and its clinical correlation).



**Figure 3:** Sources, assessment, preventive measures during renal transplantation, and advances in preventing HLA sensitization. PRA: Panel reactive antibodies, SAB: Single antigen bead, Tx: Transplantation, HLA: Human leukocyte antigen, PIRCHE-II: Predicted Indirectly ReCognizable HLA Epitopes presented by HLA class II.

Figure 3 illustrates the various steps of HLA sensitization.

In conclusion HLA mismatch is a key factor limiting long-term graft survival. Sensitization commonly results from blood transfusions, pregnancies, miscarriages,

or prior transplantation. Preventive strategies such as minimizing transfusions, using leukodepleted blood, proper immunosuppression management in graft failure, and timely clinical interventions can reduce sensitization. Desensitization therapies offer potential benefits, while emerging molecular approaches like eplet matching may improve donor compatibility, although broader clinical experience is needed for routine use.

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